The Journal

of the

Louisiana State Medical Society

$4.00 Per Annum, 35c Per Copy
Vol. 105, No. 12

DECEMBER, 1953

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

GASTROINTESTINAL HEMORRHAGE*

PANEL I. ESOPHAGUS, STOMACH, AND DUODENUM

GORDON McHARDY, M. D.
JOHN E. BECHTOLD, M. D.
and
DONOVAN C. BROWNE, M. D.
NEW ORLEANS

This Panel shall be confined to esophageal, gastric, and duodenal bleeding. Panel II, pertaining to small bowel hemorrhage was presented at the Thirty-Fourth Annual Session of the American College of Physicians, Atlantic City, April 17, 1953, as a portion of Panel discussion III, P.47: Bleeding lesions of the gastrointestinal tract.

INCIDENCE AND ETIOLOGY:

The incidence of significant upper gastrointestinal hemorrhage is probably best estimated by an etiologic survey of hospitalized gastrointestinal patients. Bleeding so mild as not to necessitate emergency hospitalization and cases admitted merely for diagnosis are omitted from this discussion. With this concept, we tabulated a twenty year hemorrhage occurrence on two gastrointestinal services at Touro Infirmary. Multiple admissions were studied as a single case, using the last admission for conclusions. There were 303 cases studied, representing 14.2 per cent of the gastrointestinal admissions, 18.6 per cent of the peptic ulcer admissions. In an etiologic breakdown, benign peptic ulcer was the most frequent, 70.9 per cent (or 215 instances), neoplasia represented 13.2 per cent, gastritis 6.2 per cent, and esophageal varices accounted for bleeding in 5.2 per cent. (See Table 1)

It is conceivable that some of the undiagnosed cases may represent small bowel bleeding; however, 4 of these cases had hematemesis and the other 2, in a follow-up beyond two years, have not manifested bowel symptomatology.

Foreign bodies, phlegmonous gastritis, gastric lues, tuberculosis, duodenitis, cardiospasm, allergies, and drug sensitivities were not encountered as bleeding sources. However, if non-thrombocytopenic purpura be considered as of allergic origin, we did have a single such experience.

Our percentage of undiagnosed cases is remarkably low. Crohn[1] has reported on 22 instances of undetermined etiology in a series of 102 bleeding cases. Jankelson and Milner[2] depict the diagnostic difficulties encountered in a series of etiologically vague bleeders. Multiple admissions probably eliminated many diagnostic possibilities and account for the high incidence of duodenal neoplasms.

ESTIMATE OF SEVERITY

All 303 patients manifested bleeding by either hematemesis (massive) or melena (grossly tarry stools). Hematemesis accompanied melena in 101 cases in our series; a third of the patients (33.3 per cent) with severe hemorrhage showed hematemesis. These 101 cases were divided 67 to peptic ulcer (31 per cent of ulcers), 16 to esophageal varices (100 per cent of varices), 13

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*Presented at the Seventy-third annual Meeting of the Louisiana State Medical Society, New Orleans, May 8, 1953.

From the Department of Medicine, Browne-McHardy Clinic and the Gastrointestinal Services of Touro Infirmary, New Orleans.

Aided by a grant from the Medical Group Research Fund.

TABLE 1
TABULATION OF 303 CASES OF UPPER GASTROINTESTINAL BLEEDING

	Diagnosis	Location	No.	Per Cent	Medical Mortality
I.	Peptic Ulcer		215	70.9	5.5
	A. Esophageal				
	1. With hiatus hernia		2		
	2. Without hiatus hernia		4		
	3. Esophagogastric stomal		1		
	B. Gastric		36		
		In herniated portion	8		
	C. Duodenal		144		
	D. Gastrojejunal (Marginal)		20		
II.	Neoplasm		40	13.2	30.5*
	A. Gastric		28		
	1. Benign		1		
	2. Malignant		27		
	B. Duodenal		9		
	1. Benign		2		
	2. Malignant		7		
	C. Esophageal		2		
	D. Adenoma Ampulla of Vater		1		
III.	Esophageal Varices		16	5.2	56†
IV.	Gastritis		19	6.2	0
	A. Acute Erosive		5		
	B. Hypertrophic		8		
	C. Post Gastrectomy		6		
V.	Miscellaneous		7	2.3	28.5‡
	A. Duodenal Diverticulum		3		
	B. Pancreatitis		1		
	C. Gastric Mucosal Prolapse into Duodenum		1		
	D. Blood Dyscrasia		2		
VI.	Undiagnosed		6	2.0	0

*Exsanguination and inoperable but explored cases.

†Hepatic death in 3 cases, exsanguination in 6 cases.

‡Death directly attributable to blood dyscrasia in 1, and pancreatitis in second case.

to gastric neoplasm (approximately 50 per cent of gastric neoplasia), and 4 to the gastritis group. Hematemesis is considered prognostically more serious than melena.[3] (See Table 2)

Melena requires approximately 60 cubic centimeters of blood to be manifest by a tarry stool, and therefore, does not necessarily indicate extensive bleeding. This is substantiated in that only 68 (33.6 per cent) of patients manifesting solely melena displayed clinical shock. However, when hematemesis is concomitant with melena, there is justification to assume severity in that 75.2 per cent were classified as in moderate to severe shock.

It is our opinion that clinical shock or impending shock as indicated by yawning, sighing respirations, rapid thready pulse of poor volume; and drop in blood pressure is an index superior to laboratory evaluation. (See Table 3)

In our case studies the exact status of laboratory procedures cannot be evaluated in that relationship of determinations to blood replacement is not accurately indicated. Ordinarily, a blood picture with red cell count below 3,000,000, hemoglobin below 9 grams, and hematocrit less than 28 depicts severe blood loss.[4] One must not be misled by such factors as early vasoconstriction, splenic release of red cells and the

TABLE 2
ESTIMATE OF SEVERITY OF HEMORRHAGE (A)

	Total Cases		Shock		Bleeding Sources							
					Ulcer		Varices		Gastric Neoplasm		Gastritis	Others
	No.	%	No.	%								
Melena (alone)	202	66.6	68	33.6	148	69%	0	(0%)	15	(53.6%)	18 (95%)	9 (69.9%)
Hematemesis and Melena	101	33.3	76	75.2	67	31%	16	(100%)	13	(46.4%)	1 (5%)	4 (30.1%)

TABLE 3
ESTIMATE OF SEVERITY OF HEMORRHAGE (B)

Laboratory Procedures:	Blood Picture (RBC)	less than 3,000,000	(Consider early (vasoconstriction,
	Hemoglobin	less than 50% or 9 gm.	(splenic liberation (of RBC, compensa-
	Hematocrit	less than 28	(tory hemodilution.
	Blood volume determinations, the cell: Plasma Relationship: Evans Blue—Radioactive Iodinated human serum albumin.		
Clinical Findings:	Significance of first hemorrhage, age, blood pressure.		
Shock Estimates:	Sacks 75% body tilt for evaluation of blood requirement.		
Physiologic Response:	Significance of vascular response to 500 cc. of blood.		

delayed compensatory hemodilution. Obviously, accurate blood volume studies would be definitive, but the Evans blue method is inaccurate and the radioactive iodinated human serum albumin determination is not generally available. Bromsulfalein liver function may evaluate hepatic status when varices are suspected. Prothrombin determinations are adjunctive. We are not impressed with conclusive significance granted progressive urea nitrogen change (which may be more of an indication of dehydration).

One cannot prognosticate on the basis of initial hemorrhage. Wilkinson and Tracey[5] feel it is not more serious than later episodes; Baker[6] assumes the first bleeding episode the source of most deaths. Age is a contributory factor in that the potential hematologic and physiologic reserves of the patient, coupled with arteriosclerosis, increase the mortality percentage. Hypertension is no longer generally conceded of prognostic significance.[7]

Sacks[8] has suggested tilting the body 75 degrees for three minutes with the following evaluative concepts:

1. If the pulse increases are less than twenty-five a minute there has been negligible blood loss.

2. If the pulse increase is more than thirty a minute, 1000 cc. blood replacement is required.

3. If syncope occurs the blood requirement is 1500 cc.

4. If shock occurs in the supine position, the need is 2000 cc. of blood.

He suggests that, if in shock, 500 cc. of blood raises the blood pressure to 100 systolic and the pulse rate concomitantly drops, that the situation is relatively safe.

In essence, the clinician evaluates the patient in terms of initial finding, response or progressive change and adapts the therapy principles next discussed to the individual case.

MANAGEMENT

It is impractical to be didactic and to anticipate in programmatic routine the management for all bleeding gastrointestinal patients. (See Table 4) Hospitalization is

TABLE 4

THE MANAGEMENT OF MASSIVE ESOPHAGO-GASTRO-DUODENAL HEMORRHAGE

1. Management of Shock and Anoxemia
   - A. Warmth
   - B. Oxygen
   - C. Sedation
   - D. Trendelenburg Position
   - E. Fluid and Blood Replacement
   - F. Control of Esophageal Bleeding
2. Patient Evaluation Within 72 Hours with Surgical Consultation
   - A. History
   - B. Examination
   - C. Laboratory Survey
   - D. Diagnostic Measures
3. Nutrition
   - A. Meulengracht Regimen
   - B. Conservative Feedings
   - C. Dietary Supplements
4. Neutralization of Digestant Pepsin and Hydrochloric Acid
5. Miscellaneous Procedures
   - A. Gelfoam and Buffered Thrombin
   - B. Sedation
   - C. Vitamin Administration
   - D. Intubation and Suction
   - E. Anti-Cholinergics
6. The Election of Sugery: "Hemorrhage Team"
   - A. Within Seventy-Two Hours (Persistent Bleeding)
   - B. Upon Recovery (Hemorrhage Controlled within 72 Hours)
   - C. Recurrent Bleeding after Initial Cessation.

imperative. Obviously, the management of shock takes precedence for one cannot estimate the potentiality of hemorrhage. Therefore, warmth, Trendelenburg position, cautious sedation, oxygen, parenteral fluids, and anticipation of blood replacement have priority over individualization.

Immediately thereafter, case requirements are analyzed. The known ulcer patient with active symptoms presents little problem. Bleeding, however, may be the initial manifestation in peptic ulcer, in neoplasia, and in varices. In a survey of 100 consecutive cases, a significant diagnostic ulcer history was elicited in 62, but was later proven erroneous in 3. Therefore, the bleeding source was not obvious from history in 41 per cent. Physical examination by virtue of a palpable gastric mass in 4 and by evidence of concomitant hepatomegaly and splenomegaly in 3 was informative in only 7 per cent.

Laboratory data indicating a blood dyscrasia was conclusive in only one instance. We did not encounter hypoprothrombinemia. Beyond this the studies were merely evaluative. Blood volumes were not done generally. Urea nitrogen in our opinion and in Crohn's is not evaluated a specific index of extensiveness of bleeding.[4]

Obviously peptic ulcer is the most likely diagnosis in approximately 80 per cent of cases, yet an error may permit procrastination in an ulcerated neoplasm or injudicious handling of a bleeding varix. Should surgery become imperative, knowledge of the ulcer site would be of estimable value. Multiple bleeding lesions are not unusual. Therefore, circumstances permitting, we, in concurrence with others, advocate the Hampton radiologic evaluation, appreciating a minimal risk. In the past 12 consecutive cases, such study has led to a conclusive radiologic diagnosis in all instances without untoward patient reaction. Elmer and his associates[10] so examined 58 cases with definitive findings in 44 cases. Crohn's[4] experience has been extensive and favorable. If the patient is not in shock or has recovered from shock, such examination is justified. We are amused to find that Meulengracht feeding advocates are violently opposed to the ingestion of a watery barium suspension.

Esophageal varices suspected by physical findings and perhaps an abnormal sulphobromphthalein retention may be radiologically demonstrable or tamponade controlled. Bleeding not controlled by the properly positioned Sengstaken tube with blood aspiratable is probably due to a gastric or duodenal lesion. Bedside esophagoscopy is reserved for the extreme emergencies. Sclerosing of varices is inferior to a balloon tamponade. Esophagoscopic sclerosing therapy, however, taught us that endoscopy was permissible during acute bleeding.

Surgical advances mitigate against the so-called conservative approach limiting thought to gastric rest, acid neutralization,

and sedation. The surgeon should be an active consultant in all bleeding cases, anticipating the need for mechanical control in some instances of massive hemorrhage and in all instances of continued of recurrent bleeding.

Tamponade control of esophageal variceal bleeding[11-15] by the triple lumen double Sengstaken tube with adjunctive thrombin permits recovery and thought of subsequent control of portal hypertension by hepatic therapy, preparation for a Blakemore portocaval shunt,[16] Garlock's mediastinal packing,[17] Crile's transesophageal venous ligation, or perhaps hepatic artery ligation. The latter procedure is not surviving well.

Blood replacement, adequate and rapid, restores blood volume, avoids anoxemia, renal and coronary insufficiency and evaluates the status of bleeding. We favor the administration of 1500 cc. of blood within the first twenty-four hours and 500 cc. every twenty-four hours thereafter to maintain a red blood cell count of 4,000,000. If the blood picture cannot be maintained by this quantity of blood, and therefore, continued bleeding exceeds seventy-two hours, surgery is advised unless other considerations exist.[9]

Pollard and Wollum do not concur in this aggressive attitude.[18] They feel liberal immediate transfusion may precipitate recurrent bleeding and a higher mortality. Dunphy and Gray[19] admonish all to differentiate chronic anemia from acute exsanguination. We cannot deny the increase in mortality since the free use of blood. Statistics, however, can depict any desired picture. We concur in demand for specific transfusion indication; no requirement could be more imperative than shock and anoxemia from blood loss, with concomitant cardiac[20] and renal insufficiencies. One anticipates clinical judgement in observance of the amount of sodium citrate and of sodium chloride administered in the course of fluid administration, and the avoidance of precipitating pulmonary edema.

Meulengracht[3, 21] has many advocates. Clinical judgement dictates the decision whether to feed the patient or to achieve gastric rest. In the first seventy-two hours after extensive blood loss the average patient is neither equal nor receptive to the task of ingesting food. Our early experience in an effort to be Meulengracht adherents found us, like Baker,[6] abandoning the effort in over a third of the cases because of recurrent emesis followed by hematemesis. Here again, case individualization dictates. Inanition is hardly important in the initial management of a patient supported parenterally. A reasonable approach is rationalized; begin to feed the patient on the cessation of bleeding, feed carefully with frequent small feedings at body temperature of bland, nonstimulating fluids and semifluids. This is simply accomplished by the ascending Sippy program or a modification thereof. Aminoacids are not impressive nor are they truly palatable. Vitamin supplements are not a part of this early program.

Neutralization of digestant pepsin and hydrochloric acid has been stressed. It seems logical to attempt such neutralization when oral feeding becomes permissable. Selection of rapidly acting absorbable antacids, the satisfactory aluminum hydroxide preparations or magnesium trisilicate is a question of patient tolerance. Some individuals complain of nausea with some preparations. Continuous drip is arduous and unnecessary.

There may be justification for a trial of oral gelfoam and buffered thrombin for topical achievement of hemostasis.[22]

Sedation, achievable parenterally by the simplest nonemetic barbiturates rather than by narcotics is preferable. Relief of apprehension is sought and not a nonresponsive patient, since in the critical period the patient should be watched closely.

Hypoprothrombinemia indicates parenteral vitamin K. The administration of vitamin B complex is justified if a period of anoxemia has potentiated hepatic insufficiency. Beyond these two, dietary supplements are adjunctive to later management.

Intubation and gastric suction are potentially dangerous. The same may be said of

diagnostic gastroscopy which we have advocated in the past. The occurrence of bleeding in patients on anticholinergic therapy, coupled with an experience in 4 consecutive cases in whom bleeding was continuous while on these agents, has led us to feel they are contraindicated. Gastric and duodenal atonicity produced by these agents may prevent muscular contraction which may be an aid to hemostasis.

Although only about 10 per cent of massive bleeding cases will require emergency surgery, the internist has an obligation to think surgically,[23] and is not transgressing on his surgical colleague when he looks for a cooperative surgeon, should his consultant be too conservative. Surgical mortalities should be reasoned only in terms of emergency surgery within seventy-two hours after the onset of bleeding, when bleeding is recurrent after initial control, or as an elective procedure when the patient has recovered from the bleeding episode. Alertness, coupled with ability, is then the answer. In our experience, obviously limited, 12 consecutive cases have been operated during the first seventy-two hours without a mortality. Five recurrent bleeders (during the initial hospital period while under treatment) have been operated within twenty-four hours without a death. Six patients with delayed surgery and continuous bleeding have been operated with 3 deaths. Crohn,[4] in an extensive critical review of the literature, garnered 805 cases operated within forty-eight hours after the onset of bleeding showing a gross average mortality of 10 per cent. He estimates a 25 per cent mortality in delayed surgery. Obviously, even under such critical circumstances, aggressiveness on the part of the internist for surgery is acceptable.

The operative procedure is the surgeon's election.

DISCUSSION

It has been aptly stated that no form of therapy prevents recurrent hemorrhage in approximately 25 per cent of patients.[5] Of 124 patients handled medically, 30 per cent bled again. Subtotal gastric resection for hemorrhage did not prevent a second episode in twenty-four per cent. This information permits one to restress patient individualization. Regardless, our initial desire is to have the patient survive the particular episode under observation. Accurate and early diagnosis, liberal blood replacement, careful nutrition and cooperative "hemorrhage teams,"[4] in our opinion, achieve an optimistic outlook for the bleeding gastrointestinal patient.

Cessation of hemorrhage within seventy-two hours is favorable. Persistent bleeding beyond this period and recurrent bleeding render the prognosis grave and indicate operative intervention.

We call attention to our single case of hemorrhage, assumed due to erosion of gastric mucosa prolapsing into the duodenum, and to similar reported instances.[24-26] It is hoped such critical evaluation[27, 28] may establish the status of this questionable entity, ameliorating enthusiastic surgical endorsement.[29]

In an analysis of the indications for operation in hiatus hernia, Sweet[30] reported blood loss in 19 per cent of 134 cases without indicating the extensiveness of bleeding. Bow and Miller[31] report 4 instances of massive hemorrhage resulting from hiatus hernia. We herewith report 10 cases with massive hemorrhage. In concurrence with Graham's[32] editorial remarks, the danger of ulceration and hemorrhage in the presence of hiatus hernia demands elective surgical repair. That one, however, can become too enthusiastically aggressive is indicated by Palmer,[33] who seemingly elects diagnostic priority to achieve an immediate surgical mortality of 26 per cent.

SUMMARY

It is our impression that a dynamic attitude toward massive upper gastrointestinal hemorrhage requires cooperation of internist, radiologist, and surgeon. Evaluation, early accurate diagnosis, adequate management with preservation of body physiology and indicated surgery are the requisites to more survivals.

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DIAGNOSIS AND TREATMENT OF COMMON LOW BACK DISORDERS*

W. R. HAMSA, M. D.†
OMAHA, NEBRASKA

Low back pain is in all likelihood one of the most frequently encountered symptoms, being rivaled by only one other bizarre symptom, namely headache. Both are legion in their causes and in their interpretations. The controversial explanations of low back pain indicate our medical shortcomings, as every specialty has failed to relieve every patient treated. It is logical to assume that the ordinary exertions of everyday life will produce strain of soft tissues, namely ligaments and muscles rather than complicated bone or nerve lesions; continued logic suggests that accurate diagnosis plus adequate treatment will effect recovery in these common backaches which the family physician encounters. An attempt will be made to present the orthopedic evaluation and treatment of common entities causing, or prone to cause, low back pain.

The development of the human embryo to birth graphically represents man's gradual transition to a biped stance. This transition occurred in the low back and in the hip joints. Requisites for this change were ex-

*Presented at the Tri-State Medical Society Meeting, Shreveport, La., Oct. 1, 1953.

†Professor of Orthopedic Surgery, University of Nebraska College of Medicine, Omaha.

tension of the lumbosacral and hip joints, strengthening of back and hip extensor muscles, and a tilt of the pelvis to an angle of forty-five of fifty degrees, known as the angle of inclination. Variation may occur in the extension of low back or hip joints with only one region contributing chiefly; this result in either a flat or in a lordotic back alignment. (Fig. 1)

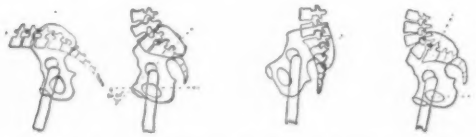

Figure 1.—Transition from quadruped to biped.

At birth, the child retains the fetal position of flexion of spine and extremities comparable to that of a quadruped. When muscle power is adequate, usually at two months, extension of the cervical spine occurs on raising his head. Similarly when standing occurs, hip extension causes the lumbar lordosis. The final result consists of cervical and lumbar extension positions, with thoracic and sacral flexion.

CLINICAL EVALUATION

Posture may be described theoretically as the relationship of the line of gravity to the center of gravity.[1] For practical clinical purposes, however, it should be considered as that state of balance and alignment that is most efficient for the body, whether at rest or in motion. Optimally, the line of gravity passes upward from a point slightly anterior to the ankle joint, through the center of knee and hip joints, crossing the spine at lumbosacral, dorsolumbar, and cervicaldorsal junctions, and ending at the mastoid process. Physiologically this relationship provides for optimal respiratory exchange and maintains abdominal viscera in place by anterior abdominal muscle tension. It is this anterior abdominal profile that will be kept in mind in the clinical evaluation of posture.

An accurate and detailed history is of extreme importance in the evaluation of a low back pain patient. Onset of symptoms in adolescence suggests congenital low back changes. Weight changes may coincide with onset of discomfort. A record of exact dates of injury and of onset of backache will prove valuable, especially in industrial injuries. General physical examination will usually bring out the possibility of pain referred from other systems such as gastrointestinal, gynecological, and urological. In problem cases, insist on consultation with a specialist in internal medicine or with a specialist in the field suspected as a cause for referred pain.

The orthopedic examination of the low back can only be performed on a patient adequately disrobed. Good visualization of the back and legs is possible only if the patient is wearing a pair of shorts; the addition of a bra for females does not materially obscure vision.

In the standing position note any list to one side in the frontal plane; usually this suggests a short leg, a scoliosis, or pressure neuritis. On lateral view, note posture changes. The round upper back and prominent abdomen are readily discernable. As a general rule a flat abdomen on lateral view suggests good muscular control; for practical purposes, if a line dropped perpendicularly downward from the upper abdomen suggests this to be the most anterior portion of the body, we can assume posture to be within normal limits. Prominence of abdomen at level of, or below, umbilicus indicates a disturbance of the mechanics of the lumbosacral joint; its normal extension has been increased to compensate for this prominence anterior to the line of gravity, a situation often painful in itself and always predisposed to strain and impingement. On the other hand, the absence of any lordosis in lumbar region, the flat back, can be visualized; this back, without curves to absorb shock is prone to ligamentous and muscular strains.

Range of motion is estimated in standing and in sitting positions. On standing erect with knees fully extended, record the degree of flexion by measuring distance between fingertips and floor if unable to bend fully; this motion is decreased in muscular and ligamentous strains. Estimate extension ability; this motion is decreased primarily in nerve or ligament impingements. Side bending causes compression on homolateral

side and tension on contralateral side; right lateral bending will invariably reproduce left sided muscular and ligamentous pain and usually reproduces right sided nerve compression pain. Rotation limitation may be definite, but this pattern is not diagnostically consistent in the low back.

In the sitting position, knee flexion releases hamstring tendons and the sciatic nerve primarily. Increase of spine flexion range in sitting position as compared to standing position suggests a nerve irritation in the lumbosacral level or a disturbance in the sacroiliac joint.

On recumbency, note if the patient allows spontaneous correction of a previously present list; this is a common occurrence in mild nerve compression syndromes. Record leg length, using the distance from the anterior superior iliac spine to internal malleolus. Similarly, record leg and thigh sizes; the presence of atrophy suggests motor weakness incidental to nerve disturbance.

A large variety of leg tests or maneuvers are available to localize the pathology by the indirect application of stresses to the low back. The commonly used tests, however, are as follows:

1. The straight leg raising test—(Goldthwaite or LaSague): In recumbent position, the leg is elevated while knee is kept fully extended and the degree of elevation allowed is recorded. The maneuver increases tension in hamstring tendons and muscles and in the sciatic nerve trunk and roots; hamstring pull on ischium flexes the lumbosacral joint as well. Pain may be reproduced in the lumbosacral joint, in the sciatic notch, or in sciatic nerve distribution on the same or opposite side.

2. The crossed leg test—(LaGuere, Patrick, Faber): In the recumbent position one lower extremity is flexed at the hip and knee, abducted at the hip by placing the ankle across front of opposite knee and backward pressure applied to the knee region. Stresses are delivered to the joints of the sacroiliac and hip of the same side. During this test prevent rotation of the pelvis by a simultaneous backward pressure on the opposite anterior superior iliac spine.

3. The sacroiliac test—(Gaenslen): In the recumbent position the patient resting close to one edge of the examining table, the hip joint is hyperextended by dropping this leg over the edge, simultaneously flexing the opposite hip and knee fully. Shearing stresses are established between the two iliac bones as the hyperextended hip side extends its ilium while flexed hip side flexes its counterpart. With the sacroiliac joints as integral parts of the pelvic ring, weakness, strain, or inflammation in either sacroiliac joint will be aggravated by this test.

4. The anterior thigh contracture test—(Ely): In the prone position one knee is flexed passively noting angle at which pelvis begins to extend on the spine. This maneuver demonstrates shortening or contracture of the anterior hip and thigh muscles, usually of congenital nature. Simultaneously, its extension of lumbosacral joint causes impingement of structures, reproducing lumbosacral pain if pathology is present. On occasion, both low back pain and referred pain may occur.

POINTS OF TENDERNESS

Points of tenderness are present only when pathology is in tissues sufficiently superficial to be compressed by the examining finger. After the patient has relaxed in the prone position, note presence of protective muscle spasm in sacrospinalis groups, then ascertain if tenderness is present over the following landmarks: (Fig. 2)

1. Gluteus maximum origins.—The commonest strain is in this mass and the middle third immediately lateral to the posterior superior iliac spine may be palpated as a tender fibrotic strand.

2. Sacrospinalis origins.—These muscles on active contraction splint the low back guarding against painful movements and prolonged spasm produces strain signs at their origins.

3. Gluteus medius and tensor fascia femoris origins.—Mass splinting of the low back may produce muscle tenderness due to overactivity.

4. Lumbosacral joints.—Due to its position above the sacrum and being mechanically fitted for the three degrees of motion,

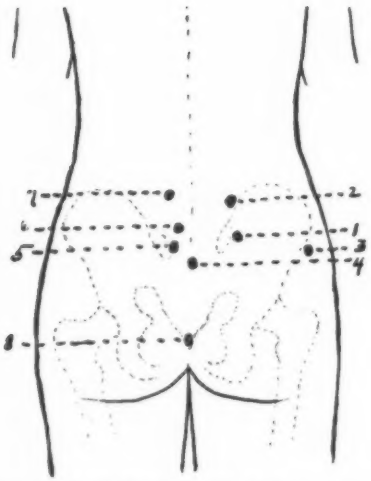

Figure 2.—Low back points of tenderness: (1) gluteus maximus; (2) sacrospinalis; (3) gluteus medius and tensor fascia femoris; (4) lumbosacral joint; (5) sacroiliac joint; (6) iliolumbar angle; (7) sacrospinalis muscle mass; (8) coccyx.

flexion extension, lateral bending, and rotation, this joint receives the major portion of all stresses. Any variation in construction predisposes to strain of its supportive ligaments.

5. The sacroiliac joint:—only the posterior ligaments of these joints are palpable posteriorly. These are located medial to the posterior superior iliac spines and are deep to the lumbodorsal fascia which is also compressed in this test.

6. The iliolumbar angle:—located between the fifth lumbar spinous process and the posterior portion of the iliac crest ending in the posterior superior iliac spine is an angle in which deep pressure may reach the transverse processes of the fifth lumbar vertebra, facets, and nerve roots. The presence of pathology about any of these structures lowers the threshhold of pressure pain.

7. Sacrospinalis muscle mass:—muscle or fascial inflammation due to any cause is invariably associated with pressure pain.

8. The coccyx:—located at the lowest portion of the spine, this structure is particularly prone to injuries; these may be single or repeated.

RECTAL EXAMINATION

Rectal examination is always necessary for orthopedic evaluation. Degenerative changes from trauma to the sacrococcygeal joint can be reproduced by passive motion of coccyx between the examining finger and thumb. Strain of gluetus maximus and of levator ani insertions can be localized. Lastly, the sacrospinous sacrotuberous ligaments, stabilizers of the sacroiliac joints may be palpated in most patients.

NEUROLOGICAL STUDY

The close association of musculoskeletal structures with spinal nerves makes a neurological study necessary. Record activity of knee and ankle reflexes. Test for sensory disturbance. A sharp pin scratch of persistent degree will demonstrate a deficiency in acuity if it is present. Lastly, test the relative muscle power of all foot and knee muscles, making comparison between the two sides.

ROENTGENOLOGICAL STUDY

Immediate roetngenological study is indicated. This includes anteroposterior, lateral, and on occasion, oblique views. A negative result gives assurance of diagnosis of soft tissue strain; conversely, the finding of bone or joint changes indicates their existence before the strain incident, especially important in any medico-legal problem. Degenerative arthritis is very common and may be expected in most individuals over fifty years of age. Common congenital changes include asymmetry of sacrolumbar facets, variations in pelvic inclination, unilateral and bilateral sacralization of fifth lumbar vertebra, pedicle defects, and spina bifida occulta. One may assume that any back with any of these changes is predisposed to strain incidents.

CLINICAL PICTURES

On the basis of the foregoing tests, a number of frequently encountered, well defined clinical pictures are noted. The prominent characteristics of each are as follows:

1. Gluteus maximum muscle strain:
   a. decreased spine flexion erect
   b. occasionally positive straight leg raising test
   c. tenderness lateral to posterior superior iliac spine

2. Sacrospinalis muscle strain:
   a. decreased spine flexion, erect or sitting
   b. Positive straight leg raising test
   c. Tenderness upper edge of iliac crest

3. Gluteus medius and tensor fascia femoris strain:
   a. shuns spine extension erect
   b. positive local pain on Ely test
   c. tenderness above or anterior to greater trochanter

4. Lumbosacral joint strain:
   a. decreased spine flexion and extension, erect or sitting
   b. positive straight leg raising bilaterally
   c. sacrospinalis muscle spasm occasionally
   d. tenderness over lumbosacral joint

5. Lumbosacral joint strain with instability:
(Potential herniated disc syndrome)
   a. marked decrease in all spine motion
   b. malposture (flexion of trunk)
   c. list to one side erect, occasionally also when recumbent
   d. pain reproduced by Ely test

6. Traumatic coccygodynia:
   a. pain reproduced on standing up from sitting position, by prolonged sitting, and during bowel movements
   b. occasionally positive straight leg raising
   c. tenderness over coccyx posteriorly and anteriorly
   d. passive coccygeal motion pain

TREATMENT

Many back strains in patients seen in office practice are of moderate severity, and following full evaluation, a therapeutic routine can usually be started without need for complete bedrest. Logical steps in this routine follow.

1. Pain Relief.—Opiates in some form are usually necessary; a combination of codeine and empirin compound adds salicylate action. Tolserol in 0.5 gram doses q.i.d. relaxes muscle spasm.

2. Physical Therapy.—Tenderness localized to muscle origin or insertion may be interpreted as a strain complex. Compression massage to such an area momentarily reproduces pain and is followed by relief. Lacking a technician, the physician may administer this treatment in a few moments. Treatments may be given daily or every two days; less frequent treatment fails to produce any cumulative effect. Diathermy heating offers relief from pain; it is used to best advantage before massage.

3. One or 2 per cent novocain injection.—Two cc. of solution placed into a muscular origin or into a ligament strain affords relief primarily and serves as a diagnostic test secondarily. More prolonged action can be obtained by using eucupin in oil in selected cases.

4. Decreased Activity.—The patient's tolerance should govern the amount of activity allowed. Pure muscular strains recover more rapidly if allowed active action plus half hour periods of rest in recumbency.

5. Support.—Adhesive plaster strapping affords quick and fairly efficient support. The addition of a commercial abdominal and low back belt decreases the strain of malposture or of the obese abdomen. Its circular constriction effects some low back stability. The lordotic or sway back is correctable to some degree and its strain definitely relieved by a three point pressure posture brace; this is made on individual order only, as good fitting is necessary for efficient action.

6. Equalization of leg length: A heel lift on the low side is used in differences under one-half inch. Greater discrepancy calls for adequate heel lift plus slightly less sole elevation in region of anterior arch, but tapered to zero at the anterior end.

7. Posture Exercises: A patient with malposture characterized by prominent abdomen, flat chest, and round, upper back, suffering additional low back strain cannot institute immediate corrective measures. However, as soon as pain is reasonably controlled, active exercise of anterior trunk and abdomen are indicated. In recumbent position on any surface, with knees and hips bent to relax iliopsoas muscle, the head and

shoulders are raised ten times, increasing by one lift daily to total of twenty-five. Posterior shoulder muscle groups respond to swimming routine, especially if the breast stroke is used. Make the patient aware of standing as tall as possible, holding the abdomen flat. Too many exercises or instructions are confusing.

8. Dietary supervision: Any excess weight is located primarily about abdomen and hips. Weight reduction will decrease the "postural drag" on the low back as well as decrease the total weight carried. Dietary deficiencies as well as avitaminosis, should be corrected if muscles and ligaments are to reach optimal states for recovery.

9. Mattress Specification: The ideal mattress supports the body in any position while maintaining relatively normal alignment. It must be firm; it must fit the patient's height and weight. The ordinary mattress may be made quite efficient by the insertion of boards on top of the bed springs. The addition of a small pillow behind the knees in recumbency adds comfort by allowing the lumbar spine to flatten moderately.

10. Chair Specification: The ideal chair has a primary lumbar support, a minimum of 105 degrees angle between trunk and thigh, a maximum seat length and seat height of sixteen inches, a few degrees of seat tilt backward to maintain proper position against the lumbar support, and has free space below the seat for placement of the feet beneath the seat in rising.[2] Usually the patient learns rapidly which type of chair is tolerated best.

A smaller group of patients suffer pain of such degree that home or office care is inadequate. This type differs only in degree; the basic muscular strain is associated with ligamentous change as well. Localization of the latter is almost invariably in the lumbosacral region; the older concept that the sacroiliac joint was the worst offender is now quite well corrected. The steps outlined for the office treatment of milder strains are continued with some steps selected for more emphasis.

Severe lumbosacral pain suggests severe strain to ligaments of this joint. Any stretch or lengthening of those ligaments holding the intervertebral disc within its normal boundaries will be followed by local edema and pain. Bulging under the normally pent-up hydraulic pressure within the disc, and further emphasized by the secondary edema, the possibility of nerve root pressure against the posterior wall of the spinal canal is readily visualized. This pathological picture is not a "herniated disc" syndrome; it merely threatens to become this entity. Early recognition of this clinical picture, particularly if seen during the first attack, may reverse the process to a degree sufficient to relieve pain and effect healing. Absolute bed rest is therefore indicated.

The regular hospital bed with head and knee elevators and with boards under the mattress serves adequately. An overhead frame is added to provide a base for a hand hold and for traction attachments. The patient is placed in recumbency with spine, hips, and knees semiflexed by raising head and knee elevators eight to ten inches. Traction of eight pounds is applied to each leg through adhesive plaster straps. Supportive slings under thighs and calves may be used to counter-balance the weight of the legs; this is known as balanced traction. Avoid traction of the legs in full extension as this position does not relax the lumbar lordosis and may increase the patient's discomfort, especially the radiating pain; should this occur, a disc protrusion must be suspected and definitive surgical treatment contemplated. Improvement, however, usually follows; within one to two days pain may be decreased sufficiently to allow the patient to turn on his side with bed flat and traction released. Low back diathermy and muscle massage may be added, followed by other previously outlined steps.

The decision as to the patient's ability to sit up must be made with care as too early stresses will repeat the strain process. A fair test of ligament healing is evidenced by the patient's ability to raise his buttocks for bed pan use; this exertion calls for sacrospinalis and gluteal active contraction

while lumbosacral lordosis is momentarily increased. A successful step of this nature suggests the addition of a corset or brace and gradual change to sitting position, followed shortly by protected walking if progress continues. Any recurrence of slight pain indicates a return to a prior phase of activity. With continued improvement, a restricted activity program should be continued for six to twelve weeks. Failure to obtain a serviceable comfortable back after six months conservative trial places the problem in the laminectomy or fusion group.

Coccygodynia, or painful coccyx, is a symptom rather than a disease entity. Located at the lowermost point of the spinal column, the coccyx is particularly exposed to injury; its joints are subject to the same disturbances as other parts of the body. A slumping, sitting posture causes direct pressure upon the coccyx; a fall backward striking the buttocks transmits similar forces. A temporary change to an extended position is necessary during childbirth. These repeated traumata are prone to cause a degenerative arthritis of the sacrococcygeal joint.[3]

Low back pain due to "coccygodynia" is assumed to be present when the findings are localized only by the coccyx; the lumbosacral region must be normal. Treatment is aimed at removing pressure in this area and stopping coccygeal motion until its muscular and ligamentous attachments and its joints have time to heal. Upright sitting, carrying weight on posterior thighs, in an upright chair relieves most cases. Constipation must be avoided or relieved. Hot Sitz baths may give relief. Digital massage of the levator ani muscles by rectal approach has been helpful. Posture training and gluteal exercises decrease the possibility of careless sitting habits and secondary traumata. Under this routine most pain syndromes in the coccygeal region gradually become quiescent in two to three months time. Recurrences, however, are common, particularly if old posture habits are acquired. Coccygectomy in selected cases only is indicated.

Pain in the anatomical location of the sacroiliac joint has all too often been considered due to a sprain or minor displacement. Structurally, this joint is extremely stable with powerful ligaments anteriorly and posteriorly and further reinforcement in the sacrospinous and sacrotuberous ligaments; true sprains or displacements of this joint are therefore rare. The probable cause of pain in this region is strain and instability of lumbosacral joint, with referred pain laterally. For definite diagnosis of sacroiliac joint strain, sprain, displacement or irritation, findings must include positive responses by reproduction of pain with the Faber crossed-leg test, Gaenslen's test, straight-leg raising test of Goldthwait, plus tenderness to pressure over posterior sacroiliac ligaments and rectally over sacrotuberous ligaments. Sacroiliac joint change is also characterized by improved spine flexion in sitting position compared to range in standing position. Pathology in sacroiliac joint causing pain is much more apt to be inflammatory rather than traumatic.

CONCLUSION

The transition from the quadruped to the biped stance has produced an inherent weakness in the low back. This weakness is normally well controlled by adequate muscle and ligament support. Mild traumata, however, often cause strain changes in these structures. Healing of these tissues may be expected if protection is adequate and prolonged, provided predisposing factors such as malposture, obesity, and congenital changes are also considered. Surgical intervention in those patients who failed to respond to conservative treatment bears promise of satisfactory results.

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DIAGNOSIS IN LOW BACK DISORDERS*

G. C. BATTALORA, M. D.
O. L. POLLINGUE, M. D.
NEW ORLEANS

Low back pain can be caused by intrinsic and extrinsic factors. Among the extrinsic causes are pelvic disorders, systemic infections, and psychogenic factors. A decision has to be made early in the examination to decide if the condition is of local or remote etiology. In the great majority of cases this can be done by recognizing the fact that cases of low back pain due to local factors have accentuation of the pain on certain postural changes. Postural changes have very little influence on the pain when it is produced by extrinsic factors.

CAUSES

Only the more common orthopedic causes can be considered at this writing. These may be divided into postural, traumatic intervertebral disc lesions, and malignancy.

Every patient with poor posture does not necessarily have low back pain. There is some added factor which upsets the equilibrium at the lumbosacral joint level to produce the pain. Examples of this would be congenital defects, such as simple laminar defects, tropism, sacralization, spondylolisthesis and development defects such as wedging of the fifth lumbar vertebra and disc with increase of the lumbosacral angle. In later life this results in localized osteoarthritic changes at the lumbosacral facets. Disc lesions are considered by some as the main cause of low back pain. There can be no question that frank herniations do produce low back pain with or without radiation. The question which only time can answer is whether simple disc degeneration causes low back pain.

Malignancy is one of the least common causes of low back pain but must always be considered.

*Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

POSTURE

The patient, who complains of low back pain, and who has poor posture, can have the pain accentuated by putting him through motions that accentuate the postural defect. These patients have little or no pain on flexion of the spine but do have pain on extension, with the pain referred to the lumbosacral junction. Postural disorders are most common in the female during the fourth and fifth decades. There is a history of low back pain, nonradiating in character, of months or years duration. It is relieved by bed rest and is accentuated by long periods of standing or ambulation. It frequently follows abdominal surgery. This abdominal surgery may have been done to relieve the low back condition. Inactivity incidental to the surgery causes increased abdominal relaxation and this predisposes to lumbosacral decompensation.

Physical findings are usually at the minimum and these may be noted on inspection. There is an accentuation of the anterior posterior curves of the spine. There is usually a full range of motion with pain on hyperextension. The abdominal musculature is relaxed. X-rays may be normal or may show one of the previously described congenital defects. They usually show an increased lumbosacral angle.

TRAUMA

Traumatic cases are usually the result of strain as in lifting with some rotatory element added. The pain as a rule is referred to the level of the lumbosacral junction. Motion is limited in all directions in the acute case. Localized tenderness is present in the region of the fifth lumbar spine. Muscle spasm is constant. X-rays may show one of the aforementioned changes or may be normal. In cases of direct trauma, a fracture may be noted. Facet fractures require x-rays of excellent quality in various planes. In the cases of acute lumbosacral strain who have poor posture, the painful phase is sometimes prolonged by the postural defect. Psychic factors are also aggravating stimuli in perpetuating the complaints. If the x-ray shows considerable

hypertrophic changes the problem of whether the arthrosis has been aggravated arises. If motion of the spine is within normal limits it is very probable that aggravation does not exist.

TYPICAL SYMPTOMS AND SIGNS

The subject of diagnosis in intervertebral disc lesions is too extensive to be considered in such a short time. A typical case usually presents recurrent low back pain and later sciatica. The pain is usually relieved by bed rest with the hips and knees flexed. Examination reveals an ipsolateral or a contralateral list. Erector spinae spasm is noted. The lumbar lordosis is diminished or reversed. Flexion of the spine is limited and if extension plus lateral bending is limited to the side of the sciatica it usually indicates a laterally placed disc herniation. If it is limited to the opposite side, the herniation is considered to be more centrally located. Straight leg raising is limited on the side of the sciatic pain. If the sciatica is produced by raising the opposite leg it indicates a very large disc hernation which extends to the midline. The ankle jerk may be absent, diminished, or be normal. Where it is affected, it usually indicates a lesion at the fifth lumbar disc. Tenderness may be a localizing sign. Hypesthesia is almost invariably present if searched for. The power in the great toe extensor may be weakened. Ordinary x-rays may be normal or may show thinning of the fifth interspace. In doubtful cases a pantopaque visualization should be done.

If a patient complains of aggravation of the low back pain or sciatica on recumbency, one should be on guard against a tumor. Common tumors of this region are chondromas, fibromas, lipomas, hemangiomas, secondary carcinoma and intradural tumors. A typical case presents low back pain with or without a history of trauma. The findings may be those of a herniated disc syndrome or of an acute lumbosacral strain. The most significant point is that the pain is usually worse at night and is not relieved by recumbency.

In the foregoing presentation an attempt has been made to rationalize diagnosis in low back disorders. In actual practice it may not be as simple as presented, although if carefully studied most cases can be fairly accurately classified.

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THE DIFFERENTIAL DIAGNOSIS OF OSTEOID OSTEOMA*

MARY SHERMAN, M. D.†
NEW ORLEANS

It has been eighteen years since Jaffe called attention to a certain bone lesion which he named osteoid osteoma. During those years enough cases have been reported so that the clinical and pathologic features have been clearly established although the etiology is still a mystery. Osteoid osteoma is common, dramatic in its manifestations and so easy to cure that the diagnosis is important. It is not difficult to suspect clinically and the microscopic picture is so characteristic that there should be no room for doubt. Yet errors do occur.

INCIDENCE

Osteoid osteoma typically occurs in young people. It appears most often in the second decade of life but it has been identified in an infant of 18 months and in a woman of 54 years. For unknown reasons it is more than twice as common in males as in females. It has been seen in practically all bones of the body except those of the skull and face but there are sites of definite predilection. Well over half the reported lesions have been in the lower extremity, most commonly in the tibia and the femur. The small bones of the feet, particularly the astragalus, are the next most frequently involved. Only one-fifth of the lesions occurred in the upper extremity, with the majority in the humerus and the small bones of the hand. About one-tenth appeared in the spine, where the usual site is the lamina just adjacent to the pedicle. The remainder were scattered in the ribs and pelvis.

*Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

†From the Departments of Orthopedics, Ochsner Clinic and Tulane University, School of Medicine, New Orleans.

CLINICAL MANIFESTATIONS

Osteoid osteoma is always a single process which never attains a size much greater than one centimeter in diameter. Its manifestations are entirely local and the presence of systemic changes practically rules out the diagnosis. The presenting complaint is usually pain. At first the discomfort is mild and intermittent, but it gradually becomes more severe and more constant. Pain is accentuated by exercise and is typically worse at night when it prevents the patient from sleeping. As it becomes more severe, it also becomes more localized. The site of involvement is easily identified because the area is exquisitely tender and so sharply localized that it can often be covered by a finger tip.

Confusion sometimes arises because of the reactive phenomena excited by an osteoid osteoma. As in other disorders, pain is sometimes referred to distant sites. This is particularly true with involvement about the hip joint, which often causes pain in the knee. When osteoid osteoma is near a joint (or actually within it as may be the case when the femoral neck is affected) there is a secondary reaction in the joint. The synovial membrane becomes acutely inflamed, there is an accumulation of fibrin-flecked fluid, and the picture may resemble a primary arthritic process. All these manifestations subside as soon as the osteoid nidus is removed, and the condition of the joint returns to normal. If the bone focus is not removed promptly, so that inflammation persists for a long time, the joint may become permanently damaged so that hypertrophic arthritis develops.

ROENTGENOGRAPHY

The fully developed osteoid osteoma presents a characteristic roentgenographic picture. There is a small round or oval area of decreased density which corresponds to the active nidus. If there is ossification at the center, a tiny dense shadow may be seen within the nidus. Usually the thick dense shadow of sclerotic regional bone surrounds it. The amount of bony reaction varies with the location of the nidus. It is least in superficial lesions and those in cancellous bone. When the nidus is at or in the cortex, the amount of new bone formed may be so extensive and dense as almost to obscure the nidus.

TREATMENT

Osteoid osteoma is one of the most satisfying disorders to treat. Surgical removal of the nidus results in such prompt and dramatic relief from pain that the patients do not even complain of postoperative discomfort. If the nidus is completely removed, it will not recur. It is neither necessary nor desirable to remove the reactive bone. In young patients this bone undergoes complete reorganization and resorption; in older patients it may persist, but it causes no trouble.

HISTOLOGY

The microscopic picture is unmistakable. There is a loose background of vascular fibrous tissue containing variable numbers of multinucleated cells. In this background arises an irregular mass of patternless osteoid trabeculae. In the more mature portions of the nidus, some of the trabeculae will have progressed to immature bone. In the reactive bone about the nidus the marrow spaces are filled with fibrous tissue which may or may not exhibit a few elements of chronic inflammation.

REPORT OF CASES

The following cases are presented to illustrate some of the commoner errors which one can hope to avoid.

Case No. 1: A man, 34 years old, had had vague aching in the left knee and thigh for four months. He had been taking 100 gr. of aspirin daily as well as other drugs. Physical examination revealed nothing abnormal but the roentgenogram showed a small area of increased density in the lower femur (Fig. 1). This had the typical appearance of a bone island but in spite of this, and the lack of characteristic history and physical findings, a diagnosis of osteoid osteoma was made and bloc excision carried out. Not only was the pain unaffected, but three weeks after operation the patient sustained a pathologic fracture through the operative site. First a cast and then an external splint were employed. Three months after operation, when the patient was still taking large quantities of aspirin, the correct diagnosis of psychoneurosis was made. Microscopic section of the "lesion" revealed only local condensation of normal cortical bone (Fig. 2).

Fig. 1: (Case 1)—Roentgenogram shows small regular area of increased density in anterior portion lower femur. Note lack of regional reaction. This is the characteristic appearance of a bone island.

Fig. 2: (Case 1)—Photomicrograph (X 40) shows perfectly normal mature dense bone continuous with the normal cortex above.

Case No. 2: A 15-year-old boy had complained for one year of pain and swelling of the left ankle. Both symptoms were aggravated by activity. Physical examination revealed nonpitting swelling of the ankle, restricted motion, and tenderness to pressure just lateral to the midline at the joint. The patient's mother, father and brother had active pulmonary tuberculosis to which the boy had had prolonged exposure. The result of Mantoux test, however, was negative and the patient had no systemic manifestations of tuberculosis. The roentgenogram showed an oval area of decreased density wtih a denser center in the neck of the astragalus (Fig. 3). This was interpreted as a tuberculous sequestrum although the joint itself seemed well preserved The ankle was immobilized in plaster, which diminished but did not abate the pain, and streptomycin was given for three months. Twenty-one months after onset of symptoms, the boy was readmitted to the hospital with complaints and physical findings unchanged. The roentgenogram (Fig. 4) showed the joint still to be unaffected and

Fig. 3 (Case 2)—Initial roentgenogram shows no changes in the ankle. There is an oval area of decreased density in the neck of the astragalus.

Fig. 4: (Case 2)—Roentgenogram made five months later shows clearly defined oval lesion, with a dense center, a "collar" of reduced density, and sclerosis of the regional bone.

the "sequestrum" not much larger but more evident. This time the correct diagnosis was suspected and operation performed. The synovial membrane was inflamed and articular fluid increased in amount. An "irregular granular area" was removed from the neck of the astragalus. Culture and guinea pig inoculation produced no growth. The patient's symptoms were at once relieved. Microscopic examination showed the typical osteoid masses in a vascular fibrous background (Fig. 5).

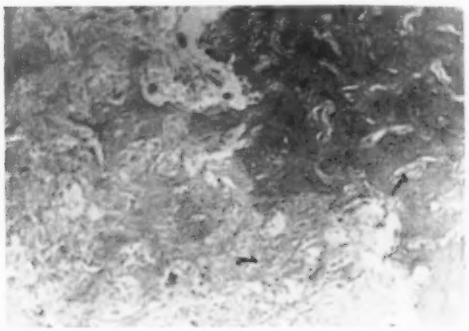

Fig. 5: (Case 2)—Photomicrograph (X 80) showing on the left the vascular fibrous tissue background with irregular masses of osteoid and on the right the bony center.

Case No. 3: A 19-year-old woman was seen because of a lump which had been present above and behind the left ear for three or four years. She was not sure whether it had increased in size. It had recently become slightly tender and vague parietal headaches developed. The roentgenogram showed a defect in the temporoparietal region with little radiating spicules of new bone (Fig. 6). Excision and repair with a tantalum plate effected a cure. The microscopic diagnosis of osteoid osteoma was made, apparently because of the large amount of new bone. However, examination showed the center of the lesion to be composed of large irregular blood spaces (Fig. 7). It was a cavernous hemangioma, a process which characteristically excites a great amount of reactive bone.

Case No. 4:* A 12-year-old girl had had pain in the low back for several months. It had gradually become severe enough to interfere with sleep. Fixed scoliosis appeared, and soon muscle spasm and acutely localized tenderness just to the right of the second lumbar spinous process became evident. When pain in the right lower extremity, weakness and a limp developed, it was realized that something more than idiopathic scoliosis was present. One and one-half years after the initial symptoms, an oblique roentgenogram revealed a large defect

Fig. 6: (Case 3)—Roentgenogram shows defect in right temporoparietal region with the fuzzy shadow of new bone at upper margin.

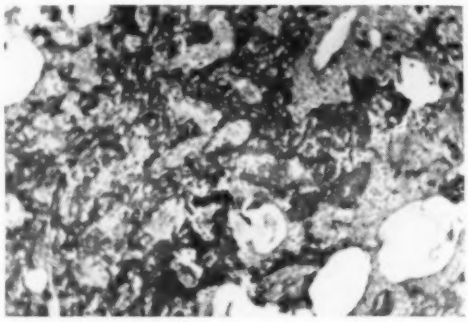

Fig. 7: (Case 3)—Photomicrograph (X 80) shows many large blood spaces surrounded by an irregular lacework of new bone and sheets of macrophages.

in the right lamina of the second lumbar spinous process (Fig. 8), excision of which relieved both the pain and scoliosis. Microscopic examination showed a perfectly typical, active, relatively immature osteoid osteoma (Fig. 9).

Case No. 5: A 43-year-old physician had vague pain and tenderness over the left tenth rib for five weeks. When a hard mass appeared at the site, he became alarmed. This mass was fixed to the rib, fusiform in shape, and nontender. The roentgeno-

*This case has been previously reported (Sherman, Mary S. and Caldwell, Guy: Osteoid osteoma. In press).

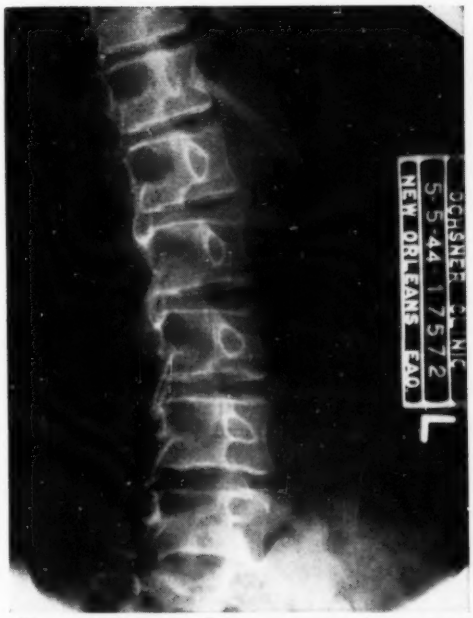

Fig. 8: (Case 4)—Roentgenogram demonstrates enlarged and thickened lamina of second lumbar vertebra with radiolucent area in center.

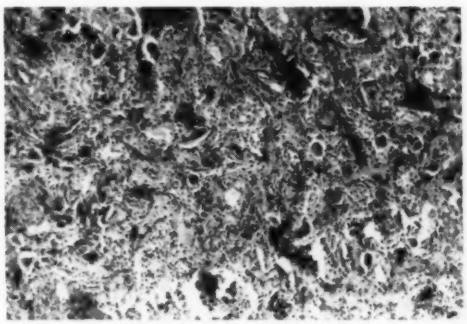

Fig. 9: (Case 4)—Photomicrograph (X 80) shows classical osteoid osteoma. Note extremely vascular fibrous stroma which contains numerous multinucleated cells. In this stroma is a network of osteoid trabeculae which in a few places have calcified to become bone.

gram showed a large smooth expansion which was decreased in density (Fig. 10). There was no shadow of reactive bone. This segment of rib was removed and the patient made an uneventful recovery. The diagnosis of osteoid osteoma was made, although nothing in the history, roentgenogram, or microscopic section suggested it. Microscopic examination showed a uniform background of fairly dense mature fibrous tissue in which slender, irregular trabeculae of immature bone arose. The rib was the site of a solitary focus of fibrous dysplasia (Fig. 11).

Fig. 10: (Case 5)—Roentgenogram (print is reversed) shows diffuse expansion of tenth rib which is decreased in density. Trabeculation is sparse but present throughout.

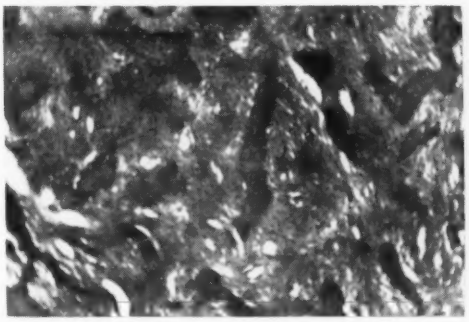

Fig. 11: (Case 5)—Photomicrograph (X 80) shows fairly mature inactive fibrous tissue in which are slender irregular trabeculae of immature new bone.

DISCUSSION

The differential diagnosis of osteoid osteoma is really simple. The errors revealed by the cases reported stem from failure to think of the diagnosis and lack of familiarity with the clinical and pathologic features of osteoid osteoma. Almost the only lesion

with which the clinical and roentgenographic picture can be confused is an indolent bone abscess but the microscopic picture is so characteristic and so constant that even this distinction offers no real difficulty.

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PITFALLS IN THE POSTREDUCTION MANAGEMENT OF FRACTURES*

NICK J. ACCARDO, M. D.
NEW ORLEANS

The subject of this paper concerns itself with those fractures treated by closed reduction methods. Often a fracture that has been properly manipulated with excellent reduction concludes with a poor functional result because of some failure in the postreduction management. This paper deals with such failures.

INADEQUATE IMMOBILIZATION

Inadequate immobilization accounts for the greatest percentage of nonunions that are found in fractures. In these times, when much pressure is being brought to bear on the doctor to return the patient to his livelihood, the question is frequently asked, even before the cast has been applied, when shall it be removed. The doctor should realize that there is only one honest answer and that is when there is x-ray evidence that the bones have united. This will take weeks and may even take months. It behooves the doctor to inform the patient at the very onset of treatment of this reality.

Immobilization has frequently been used improperly by men who certainly should know better but who, nevertheless, commit the following error. They begin their management of a fracture with one type of cast and then resort to a less formidable type of cast. For some unknown reason, the extent of the immobilization is decreased long before union has taken place. If it is sound practice to immobilize a particular fracture with a long leg plaster of Paris cast at the time of the original fracture, it is illogical, unwise, and dangerous to change to a less formidable fixation until such time as the bone fragments have completely united. All fractures, with the possible exception of fractures of the shaft of the humerus, should be immobilized by including the joints proximal and distal to the fracture site.

STABLE POSITION

A fracture that has been properly manipulated with excellent reduction obtained will redisplace unless it is immobilized in a stable position. There is only one position of stability for each fracture. Time and space do not permit a complete discussion of each and every fracture but, as a matter of example, the Colles' fracture is cited. There is only one position of stability for this fracture after it has been reduced—this position is with the wrist flexed moderately and in ulna deviation. Not to maintain this position is to cater to a redisplacement of the fragments. To exaggerate this position, as the Cotton-Loder position does, will produce swelling and venous stasis in elderly people and marked dysfunction of the hand and fingers.

On the other hand, if the position of stability of a fracture is one that places the limb in a completely unfunctional position, then it is better to do an open reduction to avoid this. An outstanding example demonstrating this problem is a fracture of the olecranon that has to be held in a reduced position by completely extending the elbow. The disability of a stiff elbow in complete extension is great.

CASTS

There are frequent errors in plaster of Paris technique that may account for an undesired result. The plaster of Paris may be put on with irregular pressure causing ridges to be formed in the cast. This source of pressure will result in soft tissue damage of varying consequence. Frequently, without being aware of it, either the assistant or the surgeon applying the plaster will produce finger grooves in the cast. This results in discomforting pressure points

*Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

and can be a source of great concern in the popliteal area. A common mistake is to apply casts which are too heavy. This will result in distraction of the bone fragments, a frequent cause of nonunion, and, in the instance of the lower extremity, will discourage ambulation. Another error of plaster of Paris technique is to attempt to move the patient too soon after the cast has been applied, before the cast sets. This causes the cast to break, requiring reapplication. A popular practice is to window a cast for frequent examination of the skin. If it is necessary that the skin be examined, it is preferable to bivalve the cast. The objection to putting a window in a cast is that it will produce localized edema and will aggravate the soft tissue damage that already exists there. Wedging of casts is a common practice today and, in the hands of a diligent, watchful operator, is good practice. It, however, can be a very dangerous procedure, as it may be the cause of pressure sores, compounding of fractures and nerve injuries. If a cast is to be wedged, the operator has an obligation to observe the patient closely for any complaints. It is improper for him to wedge a cast and to dismiss the patient from his sight not to return for weeks.

Care must be taken in applying a short leg cast with a walking attachment not to permit the attachment to extend too far below the sole of the foot. This will increase the limb length of the injured side excessively and cause the patient considerable trouble in balancing himself and in walking properly. A simple way to remedy this is to elevate the heel of the normal limb.

All casts should be trimmed properly after they are applied. No jagged, unpadded edges should be permitted to irritate the skin and produce pressure sores. A cast of the upper extremity should extend distally to the proximal palmar crease on the anterior surface of the hand to allow a full range of motion of the metacarpo-phalangeal joints and on the dorsum of the hand, it should extend distally to the end of the metacarpal heads when the metacarpo-phalangeal joints are fully flexed. To end the cast proximal to this level will produce venous engorgement and swelling of the hand and fingers.

Sometimes a patient will complain of pain after a cast has been applied. If the patient's complaints are not relieved by rearranging the pillows that support the cast, one should never resort to opiates to relieve this pain. Pain is a warning that all is not proper within the cast and to give an opiate is to disguise the true picture. Unfortunately, the pain originally complained of, if caused by pressure from the cast, may change to a numbness that will be misleading. It is only later that one learns of his error when the cast is removed and a large necrotic pressure sore with sloughing is unveiled. If any doubt exists at all that everything is not as it should be under the cast, always remove it and inspect the limb thoroughly.

Recently, I had an opportunity to observe a complication that occurred in the postreduction management of a fracture, simple, of the midshaft of the clavicle. As all of you know, there are a score or more of ways to treat this fracture—none of them are very efficient. The surgeon in this case chose to immobilize the fracture with a cotton batting figure of "8," over which was applied an Ace bandage. The patient was not seen for forty-eight hours and had developed a marked venous engorgement of both upper limbs. The reason was obvious — the figure "8" dressing was too tight about the axillae.

Improper padding of casts has resulted in many heartaches in the treatment of fractures. It is my opinion that there is no place in orthopedics for a skin tight cast in acute fracture work. The danger of vascular embarrassment is imminent and one should use proper padding to help in the absorption of the swelling that is bound to occur. One has only to realize that it is preferable to get a malunion or even a nonunion of any fracture of the upper extremity as a result of a loosely applied cast than to have the patient develop a Volkman's

ischemic contraction, a condition that defies all efforts at correction. To improperly pad a cast will result in pressure sores at the site of boney prominences. This serves as a very discomforting complication to the patient and a very embarrassing one to the physician, all of which is avoidable by simply using a little felt padding at these sites. Occasionally, however, an excessive amount of padding may result in too much play between the extremity and the cast, causing angulation of the bone fragments and, sometimes, displacement and overriding.

Improper choice of fixation and inadequate follow-up after the reduction of the fracture have been causes of illgotten results. Frequently, properly fitting casts for fractures of the forearm will become inadequate after a week of immobilization due to the atrophy of the soft tissues. If this cast is not changed and this condition remedied, the position of the bone fragments might very well change to an unsatisfactory one. It is my feeling that the ideal type of fixation of these types of fractures is the use of the sugar tong splint, which is essentially a well molded anterior and posterior layer of plaster held together by a roll of gauze bandage. This will permit the surgeon simply to cut the bandage between the two layers to release the tension if the swelling is too great and will, also, permit him to approximate the layers of plaster when atrophy of soft tissues occurs. Therefore, without jeopardizing the position of the bone fragments, constant pressure is maintained as well as maintenance of the reduction. It is my belief that women, particularly those who are obese, do not lend themselves to immobilization in plaster of Paris body casts for fractures of the spine. What is obviously a snug fitting, satisfactory cast today will be a loose fitting, inadequate cast tomorrow. I prefer to prescribe a wide brassiere and some type of corset fixation and then to attach the particular type of brace felt necessary for the given fracture to the corset.

PHYSICAL THERAPY

Failure of the physician to insist that his patients conduct a satisfactory regime of physical therapy while being treated for a fracture accounts for the many discouraging results that have been obtained. From the very first time the patient is seen he should be instructed on exercising those joints of the involved extremity that are not included in the immobilization. It is the obligation of the doctor to check to make certain that the patient is exercising. Often a patient who has suffered a fracture of the forearm will end up with a frozen shoulder or stiff fingers simply because the doctor failed in his responsibility concerning this matter. Most laymen have an erroneous opinion that *all joints* of the involved extremity should not be moved and unless they are instructed to exercise the joints not immobilized they will not do so. After the removal of the cast, it is important that the patient be given physical therapy in its various forms, such as, diathermy, warm baths, whirlpool baths, massages, dependent drainage and passive support in the form of Ace bandages or elastic stockings. To assume a patient is well and has a useful limb after there is x-ray evidence of boney union and to ignore the necessary follow-up as mentioned above is, in my opinion, to render a gross disservice to the patient.

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LEG LENGTH AND WEIGHT BEARING INTERRELATIONS*

GEORGE NELSON RONSTROM, M. D.†
NEW ORLEANS

INTRODUCTION

The low back pain problem is ever with us. General practitioners of the last hundred years were plagued by it as are specialists of more recent years. It is disheartening to have to categorize a patient as a chronic backache. It is still more discouraging to know that encouragement is about all that can truthfully be prescribed. What may seem valiant attempts at treatment to one of us may seem confused activity to an-

*From the Department of Anatomy of the Louisiana State University School of Medicine.
†Associate Professor of Anatomy, Louisiana State University School of Medicine, New Orleans.

other. Treatment is not infrequently a failure. Feelings of hopelessness and distrust disturb both physician and patient.

Bipedal progression creates a lordotic curve in the small of the back. The convexity points anteriorly and concavity posteriorly. The weakest point of a curve is at the apex of its convexity. So here then, in the small of the back, is created an exceedingly vulnerable area. From the beginning of development the lumbosacral joint is relatively weak. It is easily susceptible to deviations in its vicinity. Such deviations over a period of time can cause changes in structure and pain. Just what the individual's normal curve *is* is hard to define. It loses clear cut meaning after one sees various degrees of lordotic curvature, some with and some without pain. Normalcy here seems precariously balancing against stress and strain. Lumbar intervertebral joints, the lumbosacral joint itself and right and left sacro-iliac joints are all located here. All are part of or closely related to this precariously normal low back region. What happens in the way of deformity and pain depends on stress and strain, over a period of time, put upon the region.

Posture, locomotion, upper trunk support, etc. are important mechanisms normally exerting force on the lower back. What factors make and keep or pervert the normal forces supporting these three and possibly more such mechanisms? The forces and the mechanisms are most likely one and the same factor acting on the curve. How normal force mechanisms might be converted into stress and strain producing mechanisms stimulated this investigation. Consequent later structural changes and pain were thought of as results of perverted normal force mechanisms over a period of time but not studied.

For this study posture was chosen as a common cause of chronic lumbosacral strain In years of youth and strength, posture is relatively good. In middle age, with beginning atrophy, posture is often bad. Increased lordosis, ovoid abdomen and compensatory dorsal kyphosis appear. Postural modifications of normal supportive forces of adolescence are usually asymptomatic. In middle age with its slumping and tired posture we forget caution and lift something overly heavy. Then the accumulated and changed forces suddenly revolt with pain. From then on the back becomes chronically symptomatic. The most common cause of low back pain is very likely lumbosacral stress and strain on a postural basis. Its etiology is mechanical, consisting of perverted normal forces over the younger years with the straw that broke the camel's back as some incautious episode. The complexity of the region, the multiplicity of causes of pain must be faced. Causative factors in every case should be investigated.

METHODS OF STUDY

This study concerns itself with a mechanism common to all of us, namely, posture. It takes the viewpoint that deviations of posture over a course of time can become strain producing to the low back region. Three avenues of approach were used: (1) leg length relations; (2) right and left leg weight bearing relations; and (3) correlated interrelations of numbers one and two gave pertinent information.

Leg length relations: Sixty-seven medical students, 54 males and 13 females, were measured for leg length from anterior superior iliac spines to medial maleoli. Measurements were taken on supine subjects with metrically graduated steel rod and the aid of an assistant. Three sets of measurements were made and an average taken. The students made three daily visits and were measured the same way each time. Table 1 shows the figures.

Leg weight bearing relations: The same 67 subjects were used and weighings made on each of the three aforementioned visits as follows: (1) the total body weight was recorded in round numbers; and (2) the divided body weights on right and left extremities were taken down. To get such divided body weight a pair of automatically recording weight scales were placed against one another. It was done so that the subject could comfortably stand with a foot on each. This permitted the left scale to sepa-

TABLE 1

No.	Case	Age	Sex	Height	Average length of left extremity from 3 measurements at each of 3 visits	Average length of right extremity from 3 measurements at each of 3 visits	Difference between lengths of right and left extremities	Longer extremity
				cms.	cms.	cms.	cms.	
1.	M.T.	21	M	175	85.4	86.3	.9	Right
2.	A.V.	25	M	169	85.7	86.2	.5	Right
3.	A.T.	25	M	170	88.5	87.9	.6	Left
4.	N.T.	20	M	182	90.4	91.0	.6	Right
5.	L.S.	22	M	175	90.6	90.1	.5	Left
6.	G.S.	24	M	181	94.1	93.9	.2	Left
7.	M.S.	20	M	176	93.7	93.2	.5	Left
8.	W.S.	25	M	178	91.2	90.0	1.2	Left
9.	R.S.	29	M	177	90.8	89.7	1.1	Left
10.	J.S.	27	M	175	91.6	92.4	.8	Right
11.	R.S.	24	M	174	90.3	90.3	.0	Same
12.	M.R.	28	M	186	98.4	100.2	1.8	Right
13.	M.S.	19	M	168	86.0	87.3	1.3	Right
14.	L.G.	32	F	166	84.3	85.2	.9	Right
15.	M.B.	23	F	159	81.2	80.6	.6	Left
16.	A.B.	22	F	157	83.2	82.6	.6	Left
17.	J.P.	22	F	166	87.0	86.5	.5	Left
18.	K.B.	18	M.	163	84.5	84.5	.9	Same
19.	M.M.	27	M	163	82.6	83.1	.5	Right
20.	M.G.	26	M	188	100.4	98.9	1.5	Left
21.	B.M.	20	M	180	90.8	91.5	.7	Right
22.	G.C.	22	M	165	86.3	85.6	.7	Left
23.	R.M.	30	M	174	87.3	88.5	1.2	Right
24.	H.L.	21	M	97.9	98.3	98.3	.4	Right
25.	J.B.	27	M	172	88.4	87.6	.8	Left
26.	J.H.	25	M	200	99.6	99.2	.4	Left
27.	R.C.	22	M	168	87.0	87.1	.1	Right
28.	M.K.	25	M	168	81.4	81.2	.2	Left
29.	R.D.	27	M	176	89.1	88.8	.3	Left
30.	P.B.	26	M	174	88.8	89.3	.5	Right
31.	F.B.	27	M	167	85.5	86.5	1.0	Right
32.	L.B.	23	M	182	93.1	92.6	.5	Left
33.	J.F.	28	M	184	97.5	96.4	1.1	Left
34.	C.G.	19	M	183	98.2	98.1	.1	Left
35.	M.J.	32	F	170	84.7	84.4	.3	Left
36.	R.H.	20	M	167	88.3	87.7	.6	Left
37.	J.D.	25	M	161	80.9	82.2	1.3	Right
38.	J.M.	30	M	172	88.0	87.3	.7	Left
39.	B.P.	19	F	172	88.3	87.5	.8	Left
40.	N.P.	20	F	151	77.6	78.2	.6	Right
41.	M.S.	21	F	169	84.1	82.4	1.7	Left
42.	M.L.	20	M	171	84.1	85.5	1.4	Right
43.	R.M.	22	M	175	90.1	89.4	.7	Left
44.	R.E.	20	M	187	102.4	101.5	.9	Left
45.	M.T.	23	F	166	83.2	82.3	.9	Left
46.	M.M.	21	F	158	80.8	80.4	.4	Left
47.	M.H.	25	M	166	83.1	84.2	1.1	Right
48.	J.H.	23	M	174	88.3	89.1	.8	Right
49.	J.H.	26	M	179	92.0	90.6	1.4	Left
50.	R.H.	24	M	176	92.3	91.1	1.2	Left
51.	J.J.	27	M	173	89.1	86.4	2.7	Left
52.	H.J.	26	M	180	94.8	93.8	1.0	Left
53.	F.I.	18	M	170	87.2	86.6	.6	Left
54.	P.D.	24	M	172	88.3	87.5	.8	Left
55.	R.B.	26	M	177	86.8	86.2	.6	Left
56.	F.B.	26	M	192	105.5	106.1	.6	Right
57.	R.B.	25	M	184	98.9	99.7	.8	Right
58.	T.R.	23	M	167	86.7	88.0	1.3	Right
59.	L.P.	33	M	171	84.6	84.8	.2	Right
60.	M.D.	24	F	168	89.3	89.2	.1	Left
61.	M.C.	25	F	169	86.7	86.3	.4	Left
62.	J.K.	30	M	179	90.5	92.6	2.1	Right
63.	R.L.	25	M	183	95.2	94.2	1.0	Left
64.	P.J.	19	M	180	93.5	94.5	1.0	Right
65.	A.C.	24	M	182	94.6	94.2	.4	Left
66.	H.C.	26	M	178	92.3	93.3	1.0	Right
67.	N.C.	23	F	163	84.1	83.3	.8	Left

rately record the amount of weight bearing on the left foot and extremity and likewise for the right scale and right foot. The distance between the medial malleoli was kept at 12 centimeters until a pair of weights was made for each of the 67 subjects. Then the posture on the scale was changed to one with 30 centimeters of distance between medial malleoli. A series of divided leg weight bearing values were recorded. To complete the experiment another set of values was taken with a 45 centimeter spread between medial malleoli. Table 2 shows the figures.

Relations and interrelations of leg length and weight bearing: Twenty-nine of 54 males had longer left legs and 25 had longer right legs. Eleven of 13 females had longer left legs and 2 had longer right legs.

Thirty-one males stood with more weight on the left foot in all three positions taken on adjacent scales. Twenty-three males stood with more weight on the right foot in comparable positions. Seven females stood with more weight on the right foot while 6 stood with more weight on the left.

Twenty-eight of the total group of 67 students stood with both greater weight and length on the same side. Nineteen of these were left side and 9 right side. Thirty-seven subjects stood with greater weight and length on opposite sides. Two individuals had the same length in both legs. One of the 2 stood with greater weight on the left side and the other on the right side.

DISCUSSION

The problem started out to show posture change as a possible cause of low back strain. That is, posture from standpoint of leg length was first considered. Standing posture is supported on two lower extremities by way of the small of the back. Therefore, if marked difference in leg lengths was present in a subject his posture would tend to lean toward one side or other. He would more probably be marked out for future low back trouble. It would depend on how much compensation his body built up to stress and strain of the lean, over a period of years. However, in the 67 measurings for right and left leg length difference, only 2 people had the supposedly dangerous inch of shortening. Nineteen subjects had roughly one-half inch shortening. The remaining 46 individuals had, practically speaking, negligible shortening. Their very slight leg length differences most probably would not be a ready cause of strain. Table 1 shows the amount of difference in length between extremities for each subject.

As the problem progressed, weight bearing with its distribution on right and left extremities came to be more and more considered and finally measured as possible cause of stress and strain to the low back. The recording of weight data was set down in Table 2. Let us check subject number one on this Table 2. On first visit with feet 12 cm. apart on two adjacent scales, he distributed a total body weight of 166 lbs., 73 lbs. on left foot and 93 lbs. on right foot, a 20 lb. weight difference between the two feet, the greater weight being on the right foot. On Table 1 patient number one has a right extremity of 0.9 cms. longer than the left extremity. It seems clear to say, then, that the standing posture of subject number one with feet 12 cms. apart is 20 pounds heavier on the right foot with a right extremity 0.9 cms. longer than the left. On same first visit with feet 30 cms. apart subject number one distributed total weight 166 lbs., 80 lbs. on left foot and 86 lbs. on right. In this position there is 6 lbs. greater weight on right foot, the 0.9 cms. difference in length the same. Lastly, on the first visit the feet were spread 45 cms. apart on two scales. Subject number one distributed 166 lbs. total weight 81 lbs. on left foot and 85 lbs. on right foot. Here the greater weight of 4 lbs. is on the right foot. The 0.9 cms. longer right extremity factor is the same. To complete subject number one's posture weight variations, visits numbers two and three must be checked through Table 2 towards the right in the same manner. The distributed weight amounts vary in three postures and are fairly constant throughout three visits. It appears that weight preponderance remains right sided in subject number one. His weight distribution varied from 20 to 4 pounds as the

feet were moved from near to farther apart relaxed positions on adjacent scales. Two factors emerged: the variability of the difference in the three positions on the scales revealed itself; secondly, the weight difference, although variable in amount, remained on the same side in all three positions on three visits. In checking through Table 2 it will be seen that these two factors show up.

TABLE 2

		Feet 12 cms apart on two scales			Feet 30 cms. apart on two scales			Feet 45 cms. apart on two scales				
No.	Visit	Wgt. on left foot	Wgt. on Right foot	Visit	Wgt. on left foot	Wgt. on Right foot	Visit	Wgt. on left foot	Wgt. on Right foot	Body weight	Greater weight on	Sex
1.	1.	73 lbs.—	93 lbs.	1.	80 lbs.—	86 lbs.	1.	81 lbs.—	85 lbs.	166 lbs.	Right	M
	2.	76 lbs.—	90 lbs.	2.	80 lbs.—	86 lbs.	2.	78 lbs.—	88 lbs.		foot	
	3.	76 lbs.—	90 lbs.	3.	75 lbs.—	91 lbs.	3.	78 lbs.—	88 lbs.			
2.	1.	73 lbs.—	85 lbs.	1.	73 lbs.—	85 lbs.	1.	75 lbs.—	83 lbs.	158 lbs.	Right	M
	2.	71 lbs.—	87 lbs.	2.	73 lbs.—	85 lbs.	2.	73 lbs.—	85 lbs.		foot	
	3.	70 lbs.—	88 lbs.	3.	71 lbs.—	87 lbs.	3.	69 lbs.—	89 lbs.			
3.	1.	70 lbs.—	72 lbs.	1.	70 lbs.—	72 lbs.	1.	70 lbs.—	72 lbs.	142 lbs.	Right	M
	2.	70 lbs.—	72 lbs.	2.	69 lbs.—	73 lbs.	2.	71 lbs.—	71 lbs.		foot	
	3.	70 lbs.—	72 lbs.	3.	70 lbs.—	72 lbs.	3.	69 lbs.—	73 lbs.			
4.	1.	70 lbs.—	78 lbs.	1.	70 lbs.—	78 lbs.	1.	73 lbs.—	75 lbs.	148 lbs.	Right	M
	2.	72 lbs.—	76 lbs.	2.	72 lbs.—	76 lbs.	2.	71 lbs.—	77 lbs.		foot	
	3.	73 lbs.—	75 lbs.	3.	71 lbs.—	77 lbs.	3.	70 lbs.—	78 lbs.			
5.	1.	79 lbs.—	76 lbs.	1.	78 lbs.—	77 lbs.	1.	80 lbs.—	75 lbs.	155 lbs.	Left	M
	2.	85 lbs.—	70 lbs.	2.	83 lbs.—	72 lbs.	2.	80 lbs.—	75 lbs.		foot	
	3.	83 lbs.—	72 lbs.	3.	83 lbs.—	72 lbs.	3.	85 lbs.—	70 lbs.			
6.	1.	79 lbs.—	84 lbs.	1.	75 lbs.—	88 lbs.	1.	76 lbs.—	87 lbs.	163 lbs.	Right	M
	2.	76 lbs.—	87 lbs.	2.	78 lbs.—	85 lbs.	2.	76 lbs.—	87 lbs.		foot	
	3.	75 lbs.—	88 lbs.	3.	76 lbs.—	87 lbs.	3.	79 lbs.—	84 lbs.			
7.	1.	68 lbs.—	61 lbs.	1.	67 lbs.—	62 lbs.	1.	66 lbs.—	63 lbs.	129 lbs.	Left	M
	2.	70 lbs.—	59 lbs.	2.	70 lbs.—	59 lbs.	2.	66 lbs.—	63 lbs.		foot	
	3.	67 lbs.—	62 lbs.	3.	66 lbs.—	63 lbs.	3.	66 lbs.—	63 lbs.			
8.	1.	70 lbs.—	67 lbs.	1.	70 lbs.—	67 lbs.	1.	71 lbs.—	66 lbs.	137 lbs.	Left	M
	2.	70 lbs.—	67 lbs.	2.	70 lbs.—	67 lbs.	2.	72 lbs.—	65 lbs.		foot	
	3.	71 lbs.—	66 lbs.	3.	73 lbs.—	64 lbs.	3.	73 lbs.—	64 lbs.			
9.	1.	102 lbs.—	91 lbs.	1.	110 lbs.—	83 lbs.	1.	106 lbs.—	87 lbs.	193 lbs.	Left	M
	2.	100 lbs.—	93 lbs.	2.	100 lbs.—	93 lbs.	2.	104 lbs.—	89 lbs.		foot	
	3.	102 lbs.—	91 lbs.	3.	106 lbs.—	87 lbs.	3.	106 lbs.—	87 lbs.			
10.	1.	93 lbs.—	96 lbs.	1.	90 lbs.—	99 lbs.	1.	90 lbs.—	99 lbs.	189 lbs.	Right	M
	2.	93 lbs.—	96 lbs.	2.	90 lbs.—	99 lbs.	2.	89 lbs.—	100 lbs.		foot	
	3.	91 lbs.—	98 lbs.	3.	91 lbs.—	98 lbs.	3.	89 lbs.—	100 lbs.			
11.	1.	65 lbs.—	61 lbs.	1.	64 lbs.—	62 lbs.	1.	66 lbs.—	60 lbs.	126 lbs.	Left	M
	2.	66 lbs.—	60 lbs.	2.	65 lbs.—	61 lbs.	2.	66 lbs.—	60 lbs.		foot	
	3.	65 lbs.—	61 lbs.	3.	64 lbs.—	62 lbs.	3.	66 lbs.—	60 lbs.			
12.	1.	84 lbs.—	88 lbs.	1.	85 lbs.—	87 lbs.	1.	83 lbs.—	89 lbs.	172 lbs.	Right	M
	2.	81 lbs.—	91 lbs.	2.	84 lbs.—	88 lbs.	2.	82 lbs.—	90 lbs.		foot	
	3.	82 lbs.—	90 lbs.	3.	82 lbs.—	90 lbs.	3.	81 lbs.—	91 lbs.			
13.	1.	69 lbs.—	61 lbs.	1.	72 lbs.—	58 lbs.	1.	69 lbs.—	61 lbs.	130 lbs.	Left	M
	2.	70 lbs.—	60 lbs.	2.	72 lbs.—	58 lbs.	2.	69 lbs.—	61 lbs.		foot	
	3.	72 lbs.—	58 lbs.	3.	72 lbs.—	58 lbs.	3.	69 lbs.—	61 lbs.			
14.	1.	69 lbs.—	64 lbs.	1.	70 lbs.—	63 lbs.	1.	73 lbs.—	60 lbs.	133 lbs.	Left	M
	2.	72 lbs.—	61 lbs.	2.	70 lbs.—	63 lbs.	2.	73 lbs.—	60 lbs.		foot	
	3.	70 lbs.—	63 lbs.	3.	70 lbs.—	63 lbs.	3.	73 lbs.—	60 lbs.			
15.	1.	65 lbs.—	56 lbs.	1.	68 lbs.—	53 lbs.	1.	65 lbs.—	56 lbs.	121 lbs.	Left	F
	2.	63 lbs.—	58 lbs.	2.	68 lbs.—	53 lbs.	2.	66 lbs.—	55 lbs.		foot	
	3.	62 lbs.—	59 lbs.	3.	65 lbs.—	56 lbs.	3.	66 lbs.—	55 lbs.			
16	1.	41 lbs.—	50 lbs.	1.	43 lbs.—	48 lbs.	1.	44 lbs.—	47 lbs.	91 lbs.	Right	F
	2.	41 lbs.—	50 lbs.	2.	43 lbs.—	48 lbs.	2.	44 lbs.—	47 lbs.		foot	
	3.	39 lbs.—	52 lbs.	3.	41 lbs.—	50 lbs.	3.	38 lbs.—	53 lbs.			
17.	1.	55 lbs.—	58 lbs.	1.	56 lbs.—	57 lbs.	1.	55 lbs.—	58 lbs.	113 lbs.	Right	F
	2.	55 lbs.—	58 lbs.	2.	55 lbs.—	58 lbs.	2.	56 lbs.—	57 lbs.		foot	
	3.	56 lbs.—	57 lbs.	3.	55 lbs.—	58 lbs.	3.	55 lbs.—	58 lbs.			
18.	1.	66 lbs.—	67 lbs.	1.	65 lbs.—	68 lbs.	1.	65 lbs.—	68 lbs.	133 lbs.	Right	M
	2.	66 lbs.—	67 lbs.	2.	65 lbs.—	68 lbs.	2.	65 lbs.—	68 lbs.		foot	
	3.	65 lbs.—	68 lbs.	3.	64 lbs.—	69 lbs.	3.	62 lbs.—	71 lbs.			
19.	1.	94 lbs.—	85 lbs.	1.	92 lbs.—	87 lbs.	1.	90 lbs.—	89 lbs.	179 lbs.	Left	M
	2.	92 lbs.—	87 lbs.	2.	92 lbs.—	87 lbs.	2.	90 lbs.—	89 lbs.		foot	
	3.	95 lbs.—	84 lbs.	3.	95 lbs.—	84 lbs.	3.	92 lbs.—	87 lbs.			
20.	1.	80 lbs.—	75 lbs.	1.	81 lbs.—	74 lbs.	1.	81 lbs.—	74 lbs.	155 lbs.	Left	M
	2.	81 lbs.—	74 lbs.	2.	82 lbs.—	73 lbs.	2.	85 lbs.—	70 lbs.		foot	
	3.	84 lbs.—	71 lbs.	3.	85 lbs.—	70 lbs.	3.	85 lbs.—	70 lbs.			
21.	1.	83 lbs.—	75 lbs.	1	81 lbs.—	77 lbs.	1.	80 lbs.—	78 lbs.	158 lbs.	Left	M

		Feet 12 cms. apart on two scales			Feet 30 cms. apart on two scales			Feet 45 cms. apart on two scales				
No.	Visit	Wgt. on left foot	Wgt. on Right foot	Visit	Wgt. on left foot	Wgt. on Right foot	Visit	Wgt. on left foot	Wgt. on Right foot	Body weight	Greater weight on	Sex
	2.	85 lbs.— 73 lbs.		2.	83. lbs.— 75 lbs.		2.	80 lbs.— 78 lbs.			foot	
	3.	83 lbs.— 75 lbs.		3.	83 lbs.— 75 lbs.		3.	85 lbs.— 73 lbs.				
22.	1.	59 lbs.— 70 lbs.		1.	60 lbs.— 69 lbs.		1.	62 lbs.— 67 lbs.		129 lbs.	Right	M
	2.	60 lbs.— 69 lbs.		2.	59 lbs.— 70 lbs.		2.	61 lbs.— 68 lbs.			foot	
	3.	58 lbs.— 71 lbs.		3.	61 lbs.— 68 lbs.		3.	62 lbs.— 67 lbs.				
23.	1.	86 lbs.— 80 lbs.		1.	85 lbs.— 81 lbs.		1.	84 lbs.— 82 lbs.		166 lbs.	Left	M
	2.	86 lbs.— 80 lbs.		2.	84 lbs.— 82 lbs.		2.	84 lbs.— 82 lbs.			foot	
	3.	84 lbs.— 82 lbs.		3.	84 lbs.— 82 lbs.		3.	86 lbs.— 80 lbs.				
24.	1.	95 lbs.— 82 lbs.		1.	92 lbs.— 85 lbs.		1.	94 lbs.— 83 lbs.		177 lbs.	Left	M
	2.	94 lbs.— 83 lbs.		2.	91 lbs.— 86 lbs.		2.	95 lbs.— 82 lbs.			foot	
	3.	95 lbs.— 82 lbs.		3.	91 lbs.— 86 lbs.		3.	94 lbs.— 83 lbs.				
25.	1.	76 lbs.— 72 lbs.		1.	77 lbs.— 71 lbs.		1.	76 lbs.— 72 lbs.		148 lbs.	Left	M
	2.	75 lbs.— 73 lbs.		2.	78 lbs.— 70 lbs.		2.	78 lbs.— 70 lbs.			foot	
	3.	78 lbs.— 70 lbs.		3.	78 lbs.— 70 lbs.		3.	76 lbs.— 72 lbs.				
26.	1.	94 lbs.—106 lbs.		1.	92 lbs.—108 lbs.		1.	96 lbs.—104 lbs.		200 lbs.	Right	M
	2.	90 lbs.—110 lbs.		2.	92 lbs.—108 lbs.		2.	95 lbs.—105 lbs.			foot	
	3.	95 lbs.—105 lbs.		3.	96 lbs.—104 lbs.		3.	92 lbs.—108 lbs.				
27.	1.	78 lbs.— 74 lbs.		1.	79 lbs.— 73 lbs.		1.	78 lbs.— 74 lbs.		152 lbs.	Left	M
	2.	81 lbs.— 71 lbs.		2.	82 lbs.— 70 lbs.		2.	81 lbs.— 71 lbs.			foot	
	3.	78 lbs.— 74 lbs.		3.	81 lbs.— 71 lbs.		3.	79 lbs.— 73 lbs.				
28.	1.	60 lbs.— 75 lbs.		1.	62 lbs.— 73 lbs.		1.	61 lbs.— 74 lbs.		135 lbs.	Right	M
	2.	60 lbs.— 75 lbs.		2.	65 lbs.— 70 lbs.		2.	61 lbs.— 74 lbs.			foot	
	3.	62 lbs.— 73 lbs.		3.	62 lbs.— 73 lbs.		3.	60 lbs.— 75 lbs.				
29.	1.	101 lbs.— 90 lbs.		1.	98 lbs.— 93 lbs.		1.	98 lbs.— 93 lbs.		191 lbs.	Left	M
	2.	103 lbs.— 88 lbs.		2.	97 lbs.— 94 lbs.		2.	97 lbs.— 94 lbs.			foot	
	3.	100 lbs.— 91 lbs.		3.	100 lbs.— 91 lbs.		3.	103 lbs.— 88 lbs.				
30.	1.	74 lbs.— 61 lbs.		1.	72 lbs.— 63 lbs.		1.	70 lbs.— 65 lbs.		135 lbs.	Left	M
	2.	72 lbs.— 63 lbs.		2.	72 lbs.— 63 lbs.		2.	74 lbs.— 61 lbs.			foot	
	3.	74 lbs.— 61 lbs.		3.	70 lbs.— 65 lbs.		3.	70 lbs.— 65 lbs.				
31.	1.	75 lbs.— 70 lbs.		1.	75 lbs.— 70 lbs.		1.	74 lbs.— 71 lbs.		145 lbs.	Left	M
	2.	74 lbs.— 71 lbs.		2.	76 lbs.— 69 lbs.		2.	75 lbs.— 70 lbs.			foot	
	3.	74 lbs.— 71 lbs.		3.	74 lbs.— 71 lbs.		3.	76 lbs.— 69 lbs.				
32.	1.	89 lbs.— 86 lbs.		1.	90 lbs.— 85 lbs.		1.	90 lbs.— 85 lbs.		175 lbs.	Left	M
	2.	90 lbs.— 85 lbs.		2.	89 lbs.— 86 lbs.		2.	89 lbs.— 86 lbs.			foot	
	3.	90 lbs.— 85 lbs.		3.	88 lbs.— 87 lbs.		3.	90 lbs.— 85 lbs.				
33.	1.	92 lbs.— 80 lbs.		1.	87 lbs.— 85 lbs.		1.	89 lbs— 83 lbs.		172 lbs.	Left	M
	2.	89 lbs.— 83 lbs.		2.	88 lbs.— 84 lbs.		2.	90 lbs.— 82 lbs.			foot	
	3.	90 lbs.— 82 lbs.		3.	89 lbs.— 83 lbs.		3.	89 lbs.— 83 lbs.				
34.	1.	58 lbs.— 77 lbs.		1.	65 lbs.— 70 lbs.		1.	65 lbs.— 70 lbs.		135 lbs.	Right	M
	2.	60 lbs.— 75 lbs.		2.	65 lbs.— 70 lbs.		2.	63 lbs.— 72 lbs.			foot	
	3.	58 lbs.— 77 lbs.		3.	60 lbs.— 75 lbs.		3.	62 lbs.— 73 lbs.				
35.	1.	65 lbs.— 60 lbs.		1.	65 lbs.— 60 lbs.		1.	66 lbs.— 59 lbs.		125 lbs.	Left	F
	2.	65 lbs.— 60 lbs.		2.	64 lbs.— 61 lbs.		2.	64 lbs.— 61 lbs.			foot	
	3.	64 lbs.— 61 lbs.		3.	66 lbs.— 59 lbs.		3.	67 lbs.— 58 lbs.				
36.	1.	64 lbs.— 61 lbs.		1.	70 lbs.— 55 lbs.		1.	66 lbs.— 59 lbs.		125 lbs.	Left	M
	2.	66 lbs.— 59 lbs.		2.	69 lbs.— 56 lbs.		2.	66 lbs.— 59 lbs.			foot	
	3.	64 lbs.— 61 lbs.		3.	66 lbs.— 59 lbs.		3.	69 lbs.— 56 lbs.				
37.	1.	59 lbs.— 66 lbs.		1.	61 lbs.— 64 lbs.		1.	61 lbs.— 64 lbs.		125 lbs.	Right	M
	2.	59 lbs.— 66 lbs.		2.	62 lbs.— 63 lbs.		2.	60 lbs.— 65 lbs.			foot	
	3.	60 lbs.— 65 lbs.		3.	62 lbs.— 63 lbs.		3.	62 lbs.— 63 lbs.				
38.	1.	78 lbs.— 85 lbs.		1.	72 lbs.— 91 lbs.		1.	80 lbs.— 83 lbs.		163 lbs.	Right	M
	2.	76 lbs.— 87 lbs.		2.	71 lbs.— 92 lbs.		2.	80 lbs.— 83 lbs.			foot	
	3.	76 lbs.— 87 lbs.		3.	72 lbs.— 91 lbs.		3.	71 lbs.— 92 lbs.				
39.	1.	70 lbs.— 75 lbs.		1.	71 lbs.— 74 lbs.		1.	68 lbs.— 77 lbs.		145 lbs.	Right	F
	2.	71 lbs.— 74 lbs.		2.	71 lbs.— 74 lbs.		2.	66 lbs.— 79 lbs.			foot	
	3.	68 lbs.— 77 lbs.		3.	70 lbs.— 75 lbs.		3.	71 lbs.— 74 lbs.				
40.	1.	61 lbs.— 59 lbs.		1.	64 lbs.— 56 lbs.		1.	63 lbs.— 57 lbs.		120 lbs.	Left	F
	2.	62 lbs.— 58 lbs.		2.	64 lbs.— 56 lbs.		2.	63 lbs.— 57 lbs.			foot	
	3.	64 lbs.— 56 lbs.		3.	64 lbs.— 56 lbs.		3.	61 lbs.— 59 lbs.				
41.	1.	80 lbs.— 87 lbs.		1.	78 lbs.— 89 lbs.		1.	77 lbs.— 90 lbs.		167 lbs.	Right	F
	2.	81 lbs.— 86 lbs.		2.	77 lbs.— 90 lbs.		2.	77 lbs.— 90 lbs.			foot	
	3.	79 lbs.— 88 lbs.		3.	78 lbs.— 89 lbs.		3.	75 lbs.— 92 lbs.				
42.	1.	47 lbs.— 69 lbs.		1.	54 lbs.— 62 lbs.		1.	53 lbs.— 63 lbs.		116 lbs.	Right	M
	2.	48 lbs.— 68 lbs.		2.	53 lbs.— 63 lbs.		2.	52 lbs.— 64 lbs.			foot	
	3.	50 lbs.— 66 lbs.		3.	52 lbs.— 64 lbs.		3.	52 lbs.— 64 lbs.				
43.	1.	90 lbs.— 80 lbs.		1.	88 lbs.— 82 lbs.		1.	88 lbs.— 82 lbs.		170 lbs.	Left	M
	2.	90 lbs.— 80 lbs.		2	90 lbs.— 80 lbs.		2.	88 lbs.— 82 lbs.			foot	
	3.	92 lbs.— 78 lbs.		3.	93 lbs.— 77 lbs.		3.	93 lbs.— 77 lbs.				
44.	1.	75 lbs.— 84 lbs.		1.	77 lbs.— 82 lbs.		1.	76 lbs.— 83 lbs.		159 lbs.	Right	M
	2.	74 lbs.— 85 lbs.		2.	76 lbs.— 83 lbs.		2.	75 lbs.— 84 lbs.			foot	
	3.	76 lbs.— 83 lbs.		3.	75 lbs.— 84 lbs.		3.	77 lbs.— 82 lbs.				

		Feet 12 cms. apart on two scales			Feet 30 cms. apart on two scales			Feet 45 cms. apart on two scales				
No.	Visit	Wgt. on left foot	Wgt. on Right foot	Visit	Wgt. on left foot	Wgt. on Right foot	Visit	Wgt. on left foot	Wgt. on Right foot	Body weight	Greater weight on	Sex
45.	1.	59 lbs.—	56 lbs.	1.	58 lbs.—	57 lbs.	1.	60 lbs.—	55 lbs.	115 lbs.	Left	F
	2.	59 lbs.—	56 lbs.	2.	58 lbs.—	57 lbs.	2.	60 lbs.—	55 lbs.		foot	
	3.	58 lbs.—	57 lbs.	3.	59 lbs.—	56 lbs.	3.	60 lbs.—	55 lbs.			
46.	1.	65 lbs.—	55 lbs.	1.	62 lbs.—	58 lbs.	1.	62 lbs.—	58 lbs.	120 lbs.	Left	F
	2.	68 lbs.—	52 lbs.	2.	62 lbs.—	58 lbs.	2	63 lbs.—	57 lbs.		foot	
	3.	66 lbs.—	54 lbs.	3.	62 lbs.—	58 lbs.	3.	65 lbs.—	55 lbs.			
47.	1.	65 lbs.—	51 lbs.	1.	61 lbs.—	55 lbs.	1.	60 lbs.—	56 lbs.	116 lbs.	Left	M
	2.	66 lbs.—	50 lbs.	2.	61 lbs.—	55 lbs.	2.	60 lbs.—	56 lbs.		foot	
	3.	66 lbs.—	50 lbs.	3.	60 lbs.—	56 lbs.	3.	61 lbs.—	55 lbs.			
48.	1.	78 lbs.—	76 lbs.	1.	78 lbs.—	76 lbs.	1.	78 lbs.—	76 lbs.	154 lbs.	Left	M
	2.	78 lbs.—	76 lbs.	2.	79 lbs.—	75 lbs.	2.	79 lbs.—	75 lbs.		foot	
	3.	79 lbs.—	75 lbs.	3.	79 lbs.—	75 lbs.	3.	78 lbs.—	76 lbs.			
49.	1.	75 lbs.—	68 lbs.	1.	75 lbs.—	68 lbs.	1.	73 lbs.—	70 lbs.	143 lbs.	Left	M
	2.	75 lbs.—	68 lbs.	2.	74 lbs.—	69 lbs.	2.	73 lbs.—	70 lbs.		foot	
	3.	73 lbs.—	70 lbs.	3.	75 lbs.—	68 lbs.	3.	73 lbs.—	70 lbs.			
50.	1.	73 lbs.—	80 lbs.	1.	76 lbs.—	77 lbs.	1.	75 lbs.—	78 lbs.	153 lbs.	Right	M
	2.	73 lbs.—	80 lbs.	2.	75 lbs.—	78 lbs.	2.	76 lbs.—	77 lbs.		foot	
	3.	71 lbs.—	82 lbs.	3.	74 lbs.—	79 lbs.	3.	72 lbs.—	81 lbs.			
51.	1.	70 lbs.—	66 lbs.	1.	71 lbs.—	65 lbs.	1.	70 lbs.—	66 lbs.	136 lbs.	Left	M
	2.	69 lbs.—	67 lbs.	2.	71 lbs.—	65 lbs.	2.	70 lbs.—	66 lbs.		foot	
	3.	70 lbs.—	66 lbs.	3.	72 lbs.—	64 lbs.	3.	70 lbs.—	66 lbs.			
52.	1.	68 lbs.—	77 lbs.	1.	68 lbs.—	77 lbs.	1.	72 lbs.—	73 lbs.	145 lbs.	Right	M
	2.	67 lbs.—	78 lbs.	2.	70 lbs.—	75 lbs.	2.	72 lbs.—	73 lbs.		foot	
	3.	66 lbs.—	79 lbs.	3.	69 lbs.—	76 lbs.	3.	71 lbs.—	74 lbs.			
53.	1.	71 lbs.—	84 lbs.	1.	70 lbs.—	85 lbs.	1.	71 lbs.—	84 lbs.	155 lbs.	Right	M
	2.	71 lbs.—	84 lbs.	2.	71 lbs.—	84 lbs.	2.	70 lbs.—	85 lbs.		foot	
	3.	70 lbs.—	85 lbs.	3.	71 lbs.—	84 lbs.	3.	70 lbs.—	85 lbs.			
54.	1.	70 lbs.—	60 lbs.	1.	70 lbs.—	60 lbs.	1.	68 lbs.—	62 lbs.	130 lbs.	Left	M
	2.	70 lbs.—	60 lbs.	2.	71 lbs.—	59 lbs.	2.	70 lbs.—	60 lbs.		foot	
	3.	71 lbs.—	59 lbs.	3.	70 lbs.—	60 lbs.	3.	71 lbs.—	59 lbs.			
55.	1.	78 lbs.—	81 lbs.	1.	78 lbs.—	81 lbs.	1.	79 lbs.—	80 lbs.	159 lbs.	Right	M
	2	78 lbs.—	81 lbs.	2.	79 lbs.—	80 lbs.	2.	78 lbs.—	81 lbs.		foot	
	3.	77 lbs.—	82 lbs.	3.	78 lbs.—	81 lbs.	3.	78 lbs.—	81 lbs			
56.	1.	87 lbs.—	86 lbs.	1.	87 lbs.—	86 lbs.	1.	88 lbs.—	85 lbs.	173 lbs.	Left	M
	2.	88 lbs.—	85 lbs.	2.	88 lbs.—	85 lbs.	2.	87 lbs.—	86 lbs.		foot	
	3.	87 lbs.—	86 lbs.	3.	87 lbs.—	86 lbs.	3.	88 lbs.—	85 lbs.			
57.	1.	91 lbs.—	81 lbs.	1.	90 lbs.—	82 lbs.	1.	89 lbs.—	83 lbs.	172 lbs.	Left	M
	2.	91 lbs.—	81 lbs.	2.	90 lbs.—	82 lbs.	2.	90 lbs.—	82 lbs.		foot	
	3.	92 lbs.—	80 lbs.	3.	92 lbs.—	80 lbs.	3.	90 lbs.—	82 lbs.			
58.	1.	77 lbs.—	91 lbs.	1.	82 lbs.—	86 lbs.	1.	83 lbs.—	85 lbs.	168 lbs.	Right	M
	2.	78 lbs.—	90 lbs.	2.	80 lbs.—	88 lbs.	2.	82 lbs.—	86 lbs.		foot	
	3.	75 lbs.—	93 lbs.	3.	74 lbs.—	94 lbs.	3.	80 lbs.—	88 lbs.			
59.	1.	80 lbs.—	85 lbs.	1.	80 lbs.—	85 lbs.	1.	81 lbs.—	84 lbs.	165 lbs.	Right	M
	2.	80 lbs.—	85 lbs.	2.	77 lbs.—	88 lbs.	2.	78 lbs.—	87 lbs.		foot	
	3.	79 lbs.—	86 lbs.	3.	77 lbs.—	88 lbs.	3.	78 lbs.—	87 lbs.			
60.	1.	69 lbs.—	82 lbs.	1.	70 lbs.—	81 lbs.	1.	71 lbs.—	80 lbs.	151 lbs.	Right	F
	2.	70 lbs.—	81 lbs.	2.	69 lbs—	82 lbs.	2.	71 lbs.—	80 lbs.		foot	
	3.	68 lbs.—	83 lbs.	3.	69 lbs.—	82 lbs.	3.	71 lbs.—	80 lbs.			
61.	1.	64 lbs.—	71 lbs.	1.	66 lbs.—	69 lbs.	1.	64 lbs.—	71 lbs.	135 lbs.	Right	F
	2.	62 lbs.—	73 lbs.	2.	64 lbs.—	71 lbs.	2.	64 lbs.—	71 lbs.		foot	
	3.	61 lbs.—	74 lbs.	3.	63 lbs.—	72 lbs.	3.	64 lbs.—	71 lbs.			
62.	1.	140 lbs.—	118 lbs.	1.	133 lbs.—	125 lbs.	1.	131 lbs.—	127 lbs.	258 lbs.	Left	M
	2.	138 lbs.—	120 lbs.	2.	133 lbs.—	125 lbs.	2.	132 lbs.—	126 lbs.		foot	
	3.	141 lbs.—	117 lbs.	3.	132 lbs.—	126 lbs.	3.	134 lbs.—	124 lbs.			
63.	1.	76 lbs.—	99 lbs.	1.	83 lbs.—	92 lbs.	1.	85 lbs.—	90 lbs.	175 lbs	Right	M
	2.	78 lbs.—	97 lbs.	2.	85 lbs.—	90 lbs.	3.	85 lbs.—	90 lbs.		foot	
	3.	78 lbs.—	97 lbs.	3.	84 lbs.—	91 lbs.	3.	84 lbs.—	91 lbs.			
64.	1.	90 lbs.—	84 lbs.	1.	90 lbs.—	84 lbs.	1.	93 lbs.—	81 lbs.	174 lbs.	Left	M
	2.	90 lbs.—	84 lbs.	2.	88 lbs.—	86 lbs.	2.	90 lbs.—	84 lbs.		foot	
	3.	88 lbs.—	86 lbs.	3.	88 lbs.—	86 lbs	3.	91 lbs.—	83 lbs.			
65.	1.	85 lbs.—	78 lbs.	1.	86 lbs.—	77 lbs.	1.	85 lbs.—	78 lbs.	163 lbs.	Left	M
	2.	86 lbs.—	77 lbs.	2.	85 lbs.—	78 lbs.	2.	83 lbs.—	80 lbs.		foot	
	3.	85 lbs.—	78 lbs.	3.	86 lbs.—	77 lbs.	3.	85 lbs.—	78 lbs.			
66.	1.	76 lbs.—	71 lbs.	1.	75 lbs.—	72 lbs.	1.	75 lbs.—	72 lbs.	147 lbs.	Left	M
	2.	77 lbs.—	70 lbs.	2.	75 lbs.—	72 lbs.	2.	76 lbs.—	71 lbs.		foot	
	3.	76 lbs.—	71 lbs.	3.	76 lbs.—	71 lbs.	3.	75 lbs.—	72 lbs.			
67.	1.	56 lbs.—	63 lbs.	1.	54 lbs.—	65 lbs.	1.	55 lbs.—	64 lbs.	119 lbs.	Right	F
	2.	55 lbs.—	64 lbs.	2.	54 lbs.—	65 lbs.	2.	56 lbs.—	63 lbs.		foot	
	3.	57 lbs.—	62 lbs.	3.	54 lbs.—	65 lbs.	3.	55 lbs.—	64 lbs.			

In our group, carefully measured, the leg shortening was far below the proverbial 1 inch in most of the subjects. Yet they showed a standing distributed weight difference in three different relaxed positions on three different visits. To be sure, the amount varied but its presence remained almost constant on right or left side. Possibly this weight factor must be added and considered along with our length measuring in evaluating a patient's low back status. The author wishes to present it as such for further consideration and study by other workers. The literature, so far examined. reveals no comparable length-weight correlation study such as ours. Possibly the weight bearing difference over a period of time would act as a focus of stress and strain in individuals whose leg length difference is negligible.

SUMMARY

Leg length and weight bearing studies were made on 67 medical students. Low back pain and its cause was the objective. Variations in posture and weight bearing were sought in these young people. Such have been set down in table form as possible stress and strain factors. Distributed weight-bearing measurements related to leg length differences offer a clearer picture of possible right or left sided strain. Instances of negligible leg shortening show marked distributed weight bearing differences. The latter are offered for consideration as a focus of stress and strain on the lower back. A definite group stood with both the greater weight and greater leg length on the same side.

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FEMORAL EMBOLISM FOLLOWING INTERNAL FIXATION OF HIP*

JOHN F. NABOS, M. D.

NEW ORLEANS

With the progress of science, we practicing medicine today are seeing more and more elderly patients. It is estimated that by the end of this year there will be 13,150,-000 persons, in the United States, aged, 65 or over. This is just double the corresponding figure in 1930, and more than four times that in 1900. Today, through the combined efforts of the anesthetist, internist, and surgeon, the "old patient" has a better chance of surviving a major surgical procedure.

In the specialty of orthopedic surgery, fractures of the hip, intracapsular and intertrochanteric, are prevalent in this age group. It, therefore, seems reasonable to conclude that with the increase in the number of persons in this age group we should expect a corresponding increase in the number of patients with fractures of the hip. Recently in two consecutive admitting days, the Louisiana State University, orthopedic service, Charity Hospital, New Orleans, Louisiana, admitted 8 patients with fractures of the hip. The Tulane orthopedic service in one admitting day admitted 4 patients with fractures of the hip. All patients were in the elderly age group.

I believe it would be interesting to review the evolution of treatment of fractures of the hip. In 1902, Whitman introduced his method of reduction and external fixation. In 1925, Smith Peterson introduced open reduction and internal fixation by his now famous three flanged nail. Johansson cannulated the nail and advocated blind nailing in 1932. In the 1940's, Judet made prosthetic arthroplasty a workable procedure. Today the popular treatment of fractures of the hip, in the elderly age group, is internal fixation, and in a few selective cases of hip prosthesis.

The following report is of a complication

Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

occuring in a patient, in this age group, with a fractured hip, treated by internal fixation. It is a catastrophic vascular complication, acute occlusion of the femoral artery. It is a known fact that a larger percentage of aged surgical patients are more susceptible than young persons to postoperative vascular complications; danger increases with age.

CASE REPORT

This is a case of a white female, age 83 years, admitted February 13, 1953, with the complaint of painful left hip. She lived with a son, who was at work at the time of the accident and believed patient had sustained injury several hours before anyone discovered her.

I was requested to see the patient, by her physician, after x-rays revealed an intracapsular fracture of the left hip. He informed me patient had been under his care for arteriosclerotic heart disease, and heart failure.

On admission patient was dehydrated, febrile (101° F.), and spoke incoherently. The left lower extremity was held in external rotation and was shorter than right. Any motion caused pain. The pulse was irregular; blood pressure 170/92. The heart was enlarged and a blowing systolic murmur was heard at the apex. There were crepitant rales at the base of both lung fields. Left lower extremity was placed in Russell's traction.

Laboratory and Roentgen ray data: Blood cell count and blood chemistry were within normal limits. Urinalysis, 1 plus albumin; remainder within normal limits. The chest roentgenogram (portable) revealed calcification in the aorta with marked enlargement of the cardiac shadow. The left cardiac border extended to the lateral chest wall. The lung fields were negative for evidence of active disease. X-ray of left hip disclosed a subcapital fracture, neck left femur. Electrocardiogram taken on February 14, 1953, disclosed a left bundle branch block. Definite electrocardiographic evidence of myocardial disease. Healed anterior lateral infarct could not be ruled out.

Hospital Course: Preoperative physical survey was completed after patient was fully digitalized. On February 19, 1953, a blind Smith Peterson hip nailing was performed.

Sodium pentothal and nitrous oxide were used as anesthetic agents, Patient received 500 c.c. whole blood during surgery. Operative time one hour, thirty-five minutes. Patient was returned to ward in satisfactory condition.

On February 21, 1953, pulse irregular. A diagnosis of auricular fibrillation was made. On fourth postoperative day patient allowed up in wheel chair two hours each day. On February 24, 1953, quinidine sulfate given because of persistent auricular fibrillation.

The morning of February 26, 1953, the seventh postoperative day, patient was restless, and complained of pain in left lower extremity. I was called to see the patient when the nurse noticed that the left lower extremity was pale and cold. Patient was restless and crying out because of pain. Blood pressure, 146/86, pulse irregular. The left lower extremity was blanched, cold, pulseless, and patient was unable to move extremity. The pallor extended to mid-thigh. Diagnosis of acute occlusion of the femoral artery became evident. Lumbar sympathetic block 1-2-3-4, performed, heparin given and extremity elevated. She became more comfortable but no changes were noticed in extremity. That P.M. general condition of patient became worse. The next morning there was a line of demarcation immediately below patella. The skin below this area was now cyanotic and cold. Her general condition was worse. Prothrombin time was 70 per cent. She expired early the next morning.

The presentation of this case is a reminder to all of us of a potential complication that can and does occur in the elderly age group. It is not peculiar to the surgical case but it occurs equally as frequently in medical cases. When it does occur, it is to be treated as an emergency, be it by the conservative method or the surgical method.

DIAGNOSIS

The diagnosis of peripheral arterial embolism may be difficult and this condition can be confused with primary thrombosis of the peripheral vessel, with a propagating thrombus superimposed on a small embolus incompletely occluding the vessel, or with arterial spasm secondary to thrombosis or to a small embolus. To confirm the diagnosis the source of the embolus should be looked for. The source is almost always (1) a thrombus in the left auricular appendage due to auricular fibrillation; (2) a mural thrombus in the left ventricle as a result of coronary occlusion and myocardial infarction; or (3) thrombosis of an atherosclerotic or arteriosclerotic plaque or an aneurysm of the abdominal aorta.

The more important symptoms of peripheral arterial embolism are: pain, pallor, paralysis, absent pulsations and temperature changes. Pain is usually sudden, severe, and agonizing. However, it may not be present in all cases. In a series of 47 cases of acute occlusion of a peripheral

artery, reported by Linton only 69 per cent gave history of sudden pain.

TREATMENT

The treatment of embolus of a peripheral artery may be: (1) conservative, (2) surgical. The conservative treatment consist of paravertebral novacaine block, and/or papaverine. Linton advocates early paravertebral block to interrupt the sympathetic pathways, counteract arterial spasm, and aid in helping to establish the collateral circulation. Lesser advocates papaverine hydrochloride, intravenously, ½ to 1 grain doses, repeated at hourly intervals for the relief of arterial spasm. Intermittent venous occlusion with the oscillating bed has its supporters. Anticoagulants are used for prevention of further thrombus and to prevent any thrombus formation on the existing embolus. Allen states reduction of the prothrombin time below 50 per cent of normal is probably not safe for elderly people. When anticoagulants are given they should be continued for approximately twenty-seven days. Once a patient is given anticoagulants the paravertebral block should not be repeated because of danger of possible retroperitoneal hemorrhage.

The surgical treatment is embolectomy. Klingensmith and Theis state that the optimal conditions for favorable results are: (1) duration of the occlusion less than eight hours; (2) site of obstruction at the bifurcation of large vessel; (3) normal arterial wall at site of embolus; (4) absence of multiple emboli; (5) absence of rapid clotting tendency of the blood that will contribute to secondary or propagating thrombosis; and (6) youth of the patients, particularly those with auricular fibrillation. The operation is less likely to be successful in elderly patients with severe coronary disease.

In the prevention of this serious complication it must be realized that there is an added factor, other than those believed to be present in the production of venous thrombosis. This is a most important factor, the cardiac factor. It is advocated, by several writers that in a patient with evidence of recent or old coronary involvement, who develops auricular fibrillation, that quinidine sulfate be given in the endeavor to establish normal rhythm and to prevent thrombus formation in the auricular appendage. Anticoagulants should be given for the prevention of possible formation of mural thrombus. A recent article by Allen is recommended for those interested in postoperative venous thrombosis. The cardiac factor, in my opinion, is best managed by an internist.

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PERSONAL EXPERIENCE IN THE USE OF HOMOGENOUS AND BEEF BONE IMPLANTS;

A SUPPLEMENTARY REPORT*

S. RALPH TERHUNE, M. D.
PAUL W. SHANNON, M. D.
BIRMINGHAM, ALABAMA

The use of homogenous and heterogenous bone implantations continues "at sixes and sevens" and groups argue about their value like "Kilkenny cats." "Namby Pambys" condemn from the start and "Sir Galahads" show too much enthusiasm. Our cases are presented in the hope that they will prove valuable in eventual adjustment of reasoning, inevitably following long time balancing of multiple personal experiences. We are not "sowing dragon's teeth."

During the two years ending April 17,

*Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

1953, 130 bone implants were done on 123 patients. Refrigerated homogenous bone was exclusively employed in 90 and beef bone in 39. In one spinal fusion, we used an homogenous "H" graft at L-4 to L-5. and a beef "H" graft in the lumbosacral joint. Therefore, this report covers homogenous bone in 91 and beef bone in 40 operations.

In 4 patients, 5 procedures were supplementary additions to aid union at long bone osteotomy sites. A thirty-six years old female did not obtain union after a beef onlay across her femoral osteotomy. An homogenous onlay seven months later resulted in mature union for weight bearing after seven months.

A forty-four years old patient obtained union following a beef onlay across his femoral osteotomy. Solid union was demonstrated by manipulation under general anesthesia and by roentgenogram at fourteen weeks.

An homogenous onlay to reinforce a midtibial osteotomy in a one year old child showed union at six weeks.

The remaining case in this group was a fourteen years old boy with a midtibial shaft defect following a fracture with infection. A second stage fibular transplant to the distal tibial stump was reinforced by an homogenous onlay with screw fixation to both tibial fragments. This was done on March 20, 1953.

Since nonunion occurs so frequently after fracture in the distal third of the tibia, an homogenous cortical onlay with screw fixation was added, along with cancellous chips to a fresh comminuted fracture of the distal tibial shaft in a sixty-five year old patient on March 17, 1953.

Nonunion followed double onlay homogenous grafts with screw fixation across a congenital pseudo-arthrosis of the tibia in an eighteen months old child. The graft was repeated a year later, and solid union was present at seventeen weeks.

To substitute for the congenital absence of the distal three-fourths of a tibia in a two-year old child, the stump was attached to the fibula and the junction reinforced with an homogenous onlay. There was solid union three months later when the excess proximal part of the fibula was transplanted to form the medial portion of an ankle mortise. Subsequent slough from infection necessitated amputation.

Bank bone was employed for reinforcement in 10 attempts to reverse nonunion after long bone fractures in 9 adults. Substitution of a complete 2 inch tibial shaft defect was followed by an early uncontrollable local infection requiring midthigh amputation.

Nonunion of fractures of the midthird of the ulna and radius was corrected in 2 individuals with onlay homogenous cortical grafts and screw fixation. Mature union occurred at seven weeks in one and at eleven weeks in the other.

Three unions followed femoral shaft fracture nonunions, three, four, and six months, respectively, after open approximation of freshened fragment ends and reinforcement with homogenous cortical onlays fixed with screws.

An humeral shaft nonunion treated in a similar manner showed union at five and one-half months.

One patient suffered a refracture after the application of an homogenous graft and reinforcing plate and screws at the site of a femoral shaft fracture on resuming active ambulation at six months. An intramedullary nailing in addition to another homogenous bone graft onlay and plating was done on March 12, 1953.

A fifty-one year old woman with an excellent Smith-Peterson nailing of a well reduced femoral neck fracture on June 14, 1952, had a peg type homogenous cortical graft driven along the nail course with the addition of a Jewett nail and screw fixation on January 23, 1953, on account of slipping of the ununited fragments a few days previously during unauthorized weight bearing. A roentgenogram at eleven weeks showed weak union.

A Brittain fusion using beef cortex in a thirteen-year old child with tuberculous caries of a hip showed union at eight months.

Cortical and cancellous homogenous bone

was employed in two twelve-year old children for shelf construction to supplement acetabular roofs following poliomyelitis hip dislocations. Union was evident in both at thirteen weeks.

Consolidation of the talo-navicular and naviculo-cuneiform joints of both feet occurred in a sixteen-year old rigid flatfoot sufferer during the fifth month after inlay grafting with beef cortex.

Four benign cysts and two giant cell tumor cavities were packed with homogenous cortical and cancellous chips. Five showed complete elimination in sixty to ninety days. The other was a giant cell tumor of the distal femoral metaphysis in a twenty-five year old female. It appeared eliminated at six months. The patient returned fourteen months following the surgery because of recurrence of pain and swelling during pregnancy. Complete absorption with increase in size of the original cavity was found. Biopsy proved malignant change and upper thigh amputation followed.

One hundred spinal fusion procedures employing bank bone were done on 97 patients. These consisted of 90 cortex implantations of the Moore "clothes pin," the Breck "H," or the Rieth type joint blocks; 3 onlay sections with side to side screw fixation through spinous processes; and 7 homogenous cortical and cancellous chip additions to Hibb's fusion operations.

One of the group of 7 chip addition procedures was done fourteen months after an L-4 to S-1 "clothes pin" graft. Union had occurred in the center of this graft but there was nonunion at both ends. The patient wears a brace with no back or leg pain two months postoperatively.

Added chips were used in 4 spinal fusion operations for scoliosis correction. Union was present in each instance at eleven to twelve months.

A patient with an unstable fifth lumbar vertebra had such small lumbar spinous processes that a block graft could not be done. Chips from L-4 to the sacrum resulted in solid union at seven months.

Stabilization of a fifth lumbar spondylolisthesis was accomplished by facet joint curettement and screw fixation. Chips were added from L-4 to the sacrum. Roentgenograms indicated fusion at seven months.

An homogenous cortical onlay from L-3 to the sacrum was applied across a nonunion of a previously placed autogenous graft opposite the fifth lumbar vertebra. Roentgenograms at six months were noncommittal, but the patient was independent of external support at nine months with no subsequent back or leg pain.

Complete bilateral laminectomy to eliminate the site of a degenerative bone cyst at the third lumbar level was followed by a double cortical beef onlay from L-1 to L-5 with multiple screw fixation in a thirty-seven year old male. At eight months, union was questionable. He returned to his job as an airplane mechanic at ten months without external support. Roentgenograms at seventeen months showed apparent union with no bone cyst recurrence.

An obese twenty-six year old woman sustained a fracture-dislocation of the tenth dorsal vertebra with cord transection and complete paralysis from that level. Nursing care was facilitated by the early addition of a beef cortex section from 8-D to 1-L with multiple screw fixation. At six months there was no evidence of union but at one year union was mature. She is now being trained in crutch-aided ambulation.

We observed 70 of the 90 spinal joint block procedures for six to eighteen months. Fifty-three had follow-up roentgenograms, and 29 of these or approximately 56 per cent showed solid union six to seventeen months postoperatively. An additional 20, or 36 per cent, obtained questionable union, and the remaining 4, or 8 per cent, showed nonunion. Three of these nonunions have undergone subsequent refusion procedures.

These results are based on follow-up roentgenographic evidence only. Clinically, the results are better.

The patient who had a beef cortex block of L-4 to L-5 and an homogenous block of L-5 to 1-S presented solid union of both joints at six months. No appreciable difference was determined in the rate of union

between the beef and the human block in this case.

In one patient, an autogenous section of tibial cortex placed in the L-3 to 1-S area four and one-half years previously failed to unite. An homogenous section was applied and mature union was present at thirteen months.

An homogenous implant from L-4 to 2-S did not unite. Therefore, at fourteen months, a beef cortex section was applied. Immature union was demonstrable at seven months.

Reoentgenograms were repeated on 85 of the 130 procedures six to seventeen months postoperatively. These films include 58 homogenous and 27 beef implants.

Thirty-four, or 59 per cent, of the homogenous transplants resulted in unions while 17, or 29 per cent remained questionable, and 7, or 12 per cent represented definite nonunions.

Twenty-two of the 27 beef implants, or 81 per cent, developed union, with 3, or 12 per cent remaining questionable, and 2 or 7 per cent resulting in nonunion.

The beef implantations in this series may be "neither fish, flesh, nor good red herring," but on the other hand, they do not represent "a stalking horse."

Three homogenous and 1 of the beef implants became infected postoperatively. This rate is almost identical with the rate during the twenty-seven months preceding the existence of the bone bank, when we had 4 infections to follow 131 autogenous transplants.

It is increasingly obvious that beef bone implants are not accompanied by any greater incidence of infection.

The status of implanted bone unions in this series will of course be more accurate after repeated roentgenograms and clinical evaluation for at least two or three years postoperatively. Consequently, we insist that the present report be considered preliminary as far as beef bone is concerned, and supplementary in regard to homogenous bone implantations. At this time, it is not a report ex cathedra.

Our opinion that autogenous transplants stand the best chance for survival remains unchanged, but homogenous transplants are practical and result in a comparable number of unions. Beef implantations may not be as good as autogenous transplants but their use has been proved safe and valuable for stimulating bone union.

"In the works of man as in those of nature, it is the intention which is chiefly worth studying." (Goethe).

N. B. In our first few cases, the implanted bone was fresher and had been frozen for only a few days. Some of these patients had extremely high temperature elevations for four or five days. Subsequently, after longer periods of freezing, the patients did not have high temperature reactions postoperatively.

This same experience was encountered with the use of beef bone.

In our opinion, frozen homogenous and frozen beef bone dies after implantation and serves as a framework housing for creeping substitution. Fresh unfrozen beef bone and fresh unfrozen homogenous bone would be less acceptable by the host organism. Prolonged freezing seems to diminish the antigenic properties, and this results in fewer inflammatory reactions following implantation.

Therefore both homogenous bone and beef bone are made more acceptable for graft purposes, by prolonged freezing.

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The Journal of the Louisiana State Medical Society

Established 1844

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News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

PHYSICIANS' COOPERATION NEEDED IN STUDY OF BLOOD PRESSURE IN PERSONS OVER SIXTY-FIVE

Dr. Arthur M. Master and associates are undertaking the study of blood pressure in people 65 and over. The investigation is under the auspices of the New York Heart Association, and is being carried on with the cooperation of the National Heart Institute, and the assistance of the American Medical Association. The cooperation of physicians all over the country is being earnestly sought. They ask for very simple data on six newly examined people over 65, two of them between 65 and 69, two 70 to 74, and two 75 and over; if possible, one in the 80's. Data are requested on persons who shall be in fairly good health, up and about at the time of examination, although they may have some chronic illness. People able to work part or full time are particularly desired. If they are well at the time of examination, people with rheumatic heart disease or previous coronary disease or hypertension may be included. The most suitable subjects suggested are patients coming for routine examination, and relatives or friends, in the desired age group.

The importance of this study becomes apparent when it is realized that because of the growing number of older people in the United States, accurate information is desirable. There is a paucity of data in the age groups for this study. With the increasing life span many more millions of advanced age will be added within several decades. The thought in this study is to know (1) the normal range of blood pressure as well as the lower limit of hypertension for each age group over 65 for both sexes; (2) whether the blood pressure continues to increase with age and to vary with sex in those who are more than 65 years old; (3) whether the blood pressure is related to weight; or (4) to surface area; or (5) to height.

The relationship of hypertension to the many degenerative vascular diseases, also to hypercholesteremia and diabetes, should be re-investigated in people over 65.

This type of study was recently completed by Master and his group in 74,000 people 16 to 64 years of age. On the basis of that study new limits of normal blood pressure were proposed. From that study of people under 65, important clinical deductions have been made. Among these is the relationship between hypertension and coronary occlusion. It had been generally accepted that an etiologic relationship existed here in both sexes. However, using the limits of normal blood pressure newly established for those under 65, it was shown that such a causal relationship exists only in women; in men, no important relationship between hypertension and coronary occlusion was found.

Also, it was noted that enlargement of the heart occurred not uncommonly in men with coronary occlusion when their blood pressure had been normal. It was concluded that coronary artery disease alone could produce cardiac enlargement in men. Women, on the contrary, rarely developed cardiac enlargement if their blood pressure was normal. Consequently, hypertension appeared to be a very important factor in the production of enlargement of the heart in women.

In recent decades, the figures of 150/90 were used as the upper limit of normal for all adults over 40. This did not take into account the important influence of age by decade, and sex, on the normal blood pressure. This resulted in an excessive incidence of hypertension. The ranges of normal blood pressure for each decade and sex were published by Master in 1950. The upper limit of normal was higher for each age group than that previously used. In the study referred to an adequate source material was available among the larger industrial concerns. In the study now proposed in the older age group, it is obvious that data can only be obtained from the practicing physician.

The data needed in completing the questionnaire which will be furnished are simple. The information gained may be most important. As most of the physicians whose assistance is sought are hoping to make at least a brief excursion into the decades under investigation the medical observations ultimately to be published would be of importance to them. Those who are willing to make these simple reports for an important study should send a postcard to "Blood Pressure Study," 11 East 100th Street, New York 29, New York. The questionnaire and explanations will be forwarded to them.

The value of the study is dependent upon its breadth and accuracy. It is earnestly hoped that the profession will make these data available.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

SCHOOL HEALTH PROGRAM

Following is a report submitted by Dr. William E. Barker, Jr., who attended the Conference of Physicians and Schools held at Highland Park, Illinois, September 30 to October 2.

Nearly two hundred physicians, educators and Public Health workers participated in the Conference. The calibre of the men and women present was of the highest. The assembly was addressed by both Dr. Ed. J. McCormick and Dr. George F. Lull among other notables.

Those attending were divided into twelve groups of eighteen to twenty people, each group studying a different phase of the School Health Program. I was placed in the "Medical Society School Health Committee" group. This group was composed of eighteen men and women representing Medicine, Health and Schools.

A lively discussion was engaged in at each point as the proposed outline was spelled out in detail to the end that a quite satisfactory program was mapped out as a guide to medical societies in forming and operating a School Health Program.

The Conference was of the nature of a National Planning Group, and the outlines were made flexible enough to be of service in almost all parts of the Country. The following abstract of the meeting is so complete it is incorporated as part of this report.

Dr. Edward J. McCormick, Toledo, Ohio, President of the American Medical Associa-

tion, reported that accidents were the greatest hazard of childhood, killing more than 14,000 children annually.

He pointed out that the combined efforts of medical and lay groups already have been rewarded by reducing child mortality more than 50 per cent in the last 15 years. He predicted a further decrease. An increased control of disease not only has raised the average life span to 68 years but has kept intact many homes.

The Conference was divided into subject groups which discussed particular problems informally. On the final day, each division made its report at the general session.

The importance of adequate screening of candidates for teaching positions was emphasized by a discussion group presided over by Dr. Leland M. Corliss, Director of Health Services in the Denver public schools. The group also pointed out the need for a freer exchange of health information by schools, families and private doctors so as to assist the teacher in the proper management of her pupils. Nurses serving high schools, it was reported by the group, are valuable as health counselors and should have the same status as faculty members. The assignment of nurses to regular teaching was discouraged.

School health services in rural areas can be improved through the development of full-time local public health units, it was reported by a group headed by Dr. Louis K. Hundley, Pine Bluff, Arkansas. Health councils and women's auxiliaries of medical societies were suggested as helpful groups in surveying county health resources.

In communities where there is a shortage of health service personnel, it was urged that priority be given to such physical environment problems as pure drinking water, adequate waste disposal, heating, ventilation and lighting. Emphasis also should be placed on safety on playgrounds, in buildings and during transportation, it was added.

The importance of a plan for meeting emergencies of illness and injury at school was stressed. School lunchrooms, encouragement of immunization of children, and safety education were cited as worthwhile protective and preventive services.

Family responsibility for a child's health was stressed by a group presided over by Dr. Franklin M. Foote, New York, Executive Director of the National Society for the Prevention of Blindness.

It was suggested that teachers and other personnel be instructed in the early signs of a deviation from normal in child physical and mental health in order to recognize such changes. The importance of good school environments as a factor in health education was emphasized.

Since a health examination is a good health teaching experience, this should not be done on a mass basis, in the opinion of the group. The role of a consulting physician for the school to act as a leader in coordinating health activities was brought out.

For the first time the Conference considered mental health aspect of teaching in the classroom. A group discussing that question was presided over by Walter E. Hager, President of the Wilson Teachers College, Washington.

It was agreed that teachers strongly affect the mental and emotional health of children and that, for this reason, teachers must be thoroughly well adjusted persons in order to carry out proper teaching approaches. This, it was pointed out, calls for better selective processes in universities or colleges which train teachers.

The group felt that before this selective process would be successful, there is need to establish criteria that would raise the standards for selection of teachers. It was suggested that an organization, such as the National Education Association, undertake the setting up of a committee whose aim would be to establish such criteria.

Desirable health standards for sports programs in both elementary and high schools were recommended by a discussion group headed by Carl A. Troester, Jr.,

Washington, Secretary of the American Association for Health, Physical Education and Recreation. For elementary school children, sports involving severe body contact and competition beyond the community level at district, state and national levels, as well as "bowl games," should be avoided, it was urged. Such activities, in addition to potential hazards to physical and mental health, create situations in which the children may be exploited, whether intentionally or not, it was stressed.

For youths of high school age, it was pointed out, the program should recognize individual differences in age, physical ability, experience and health of the participants. It was urged that an adequate medical examination precede participation in vigorous activities and that a physician be present at all contests where the accident hazard is pronounced.

Creation of advisory school health councils as a means of best meeting the health needs of children was urged by a discussion group headed by Dr. John W. Shackelford, Oklahoma City, Director of local health service for the Oklahoma Department of Health. Such a council should be made up of representatives of all segments of the community, it was emphasized.

Community responsibility for the health of school children, in the opinion of discussants, rests primarily with the schools and the health departments of education in consultation with the medical and dental professions. Leadership, guidance and assistance to schools provided in the formation of health councils depend in a large measure on the support it receives from its member organizations.

The importance of interchange of health appraisal data on children was discussed by a group of which Miss Gertrude E. Cromwell, Supervisor of Nursing in the Denver public schools, was Chairman. Such interchange, it was suggested, should involve practicing physicians, school health workers and teaching personnel, parents, and community health agencies. The confidential nature of some of the information must be respected, it was pointed out. However, the value of such information as to a child's physical ability, his handicaps, his mental or emotional status, and his home conditions in effecting a proper adjustment in school, was emphasized. Epilepsy and heart cases were cited as examples.

Medical societies should have active health committees to coordinate the efforts of the profession in the promotion of the health of the school child, it was suggested by a group headed by Dr. John L. Reichert, Chicago, Chairman of the Chicago Medical Society's Committee on Child Health.

The functons of such a committee, it was pointed out, are (1) to encourage sanction by the medical profession of a sound school health program; (2) to assist in establishing sound policies and procedures in the health program, and (3) to develop reciprocal relations with the dental society, school system, health department, parent groups and other organizations. A medical society committee does not relieve the individual physician of responsibility for active participation in the school health affairs of his community, it was stressed. Maintenance of good health records is vital to the provision of sound health services, it was pointed out by a group presided over by Dr. Harold M. Erickson, Portland, Oregon, State Health Officer of the Oregon Board of Health. Such records, it was reported, can aid in a better understanding of the health needs of the individual child, and assist medical, educational, public health and other interested groups in their understanding of child health problems.

State and local conferences on physicians and schools have resulted in the development of effective school health programs, it was reported by a group headed by Dr. Ray O. Bjork, Helena, Montana, Vice Chairman of the Joint Committee on Health Problems in Education, a group sponsored by the American Medical Association and the National Educational Association.

Topics suggested for such state and local conferences were: control of communicable

diseases, emergency care, athletics and health, methods of health appraisal, screening technics, health services, health instruction, school health policies, the role of the physician and dentist in the health of the school age child, community resources available, current medical and educational practices, professional and legal obligations, and technics of evaluation and follow-through.

State medical associations, it was reported, have taken the initiative for many successful conferences. They have had the cooperation of educators, public health workers and other groups.

A group presided over by Dr. John D. Porterfield, Columbus, Ohio, Health Officer of the Ohio Department of Health, considered the problem of maintenance of the health of school personnel. It recommended: (1) periodic health examinations; (2) health education and development of good health patterns in teacher training institutions; (3) a healthy economic level with security and retirement; (4) healthy working and living conditions; (5) recognition and prestige, with opportunity to advance professionally; (6) availability of health counseling services.

The group also recommended that health examinations be made a condition of employment.

Though our efforts in this state have not progressed very far it was quite gratifying to note how closely our past efforts conform to the general plan as outlined at this Conference.

School Health is attracting unusual attention throughout the nation and in most cases is back in the hands of the profession where it so rightly belongs. To hold our own in this field and to prevent encroachment from other sources we should, as rapidly as is possible, expand our efforts in our own state to cover as many parishes as possible. Always remember that unless the plan is well organized and operated it is better not attempted. No plan at all is better than a poorly operated and half-functioning one sponsored by our society.

WILLIAM E. BARKER, JR., M. D.

REPORT OF REPRESENTATIVES TO SECTIONAL MEETING OF AMA COMMITTEE ON FEDERAL MEDICAL SERVICES TO VETERANS

Due to the inability of Dr. Pascal L. Danna, Chairman of the State Society Committee on Federal Medical Services, to attend the Regional meeting of the AMA held in Dallas on November 6, I attended the meeting at the request of the President of the Society and beg to report the following:

If you will refer to my report of the meeting held in Chicago during the past summer (this appeared in the October, 1953 issue of The Journal of the Louisiana State Medical Society), you will have before you the policy of the AMA in regard to medical care to veterans with non-service-connected disabilities.

The meeting in Dallas was of one-day duration and was presided over by Dr. Orr, Chairman of the Council on Veterans Affairs of the AMA and attended by Mr. Leo Brown, Director of the Department of Public Relations of the AMA, and his assistant, the three of whom acted as a panel to discuss the various subjects brought before them, which I shall review below.

There were delegates present from Louisiana, Mississippi, Arkansas, Oklahoma and Texas. All these delegates held high offices in their state societies. The purpose of these regional meetings is to present the AMA policy in regard to medical care to veterans with non-service-connected disabilities on a state level and to ask that each state society appoint committees on veterans affairs in each parish or district medical society and that these committees familiarize themselves with the policy of the AMA so that they can indoctrinate each physician in his component society so that he can talk intelligently and help sell this idea to such persons as he sees fit.

It was suggested that the doctors join the American Legion and Veterans of Foreign Wars posts and there present the doctors' side of this question; namely, that within ten years we will have a population of thirty million veterans who, if the present policy

of the Veterans Administration is carried forward, will present a socialized medicine program which has entered through the back door, so to speak. This will entail also a further load in the taxing program of the federal government to take care of these veterans and you will have half of the people being taxed for something for which they receive no benefit whatsoever. Also, if this program is carried forward it will mean the draining of professional and allied help into veterans hospitals, leaving a void in the private and state institutions in this country. Therefore, the people who are paying for this program will at the same time be suffering from lack of medical care themselves due to the monstrosity into which the VA would have grown. As an example, the slow turn-over of beds in VA hospitals, as compared to beds in private hospitals; a VA hospital bed is used only 7 times each year while that of a private institution is used 34 times each year by different patients.

It was suggested that the House of Delegates of each state society act on this matter and let the House of Delegates of the AMA know their stand at the Annual Meeting in June, 1954.

After this educational program has taken place, which will occupy most of the year 1954, we hope to have created so much interest that it will be possible to have the law changed in regard to veterans with non-service-connected disabilities. This may be done in one of several ways. If a doctor knows a United States Congressman or Senator let him contact him personally and explain in detail the AMA's position in this matter; have liaison committees meet with the medical societies and the veterans organizations to explain this program to the individual veteran.

It is hoped that the Hoover Commission will report favorably for the AMA on this idea within the next six to eight months. If it does, we shall lend it all our support.

You have no doubt noticed in the last week or ten days press releases from Washington in regard to a mild change in attitude by the VA in admitting patients to their hospitals with non-service-connected disabilities in that they are now asking them the following five questions:

1. What is the current value of your property, real and personal?
2. What is the current amount of your ready assets in the form of cash, bank deposits, savings bonds, etc?
3. If you own real property, what is the approximate amount of the unpaid mortgage or other indebtedness?
4. What are your average monthly expenditures, including mortgage payments and all other personal expenses, including your expenses for dependents?
5. What was your average monthly income for the last six months, from all sources?

This was discussed in detail at the meeting and it was the consensus of the representatives of the AMA present at this meeting that this was just a smoke screen thrown up by the VA and in a practical way means nothing in that the veteran is not required to take an oath and that the only person who could act on falsification of these questions is the superintendent of each individual VA hospital. They have no social service setup to follow through on these questionnaires and it therefore appears to be just something to befog the issue.

In conclusion, it was stressed that the AMA wants the veterans with service-connected-disability to receive the very finest of medical care.

Respectfully submitted,

W. Robyn Hardy, M. D.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Rapides	Second Monday of every month	New Orleans
Sabine	First Thursday of every month	Monroe
Second District	First Monday of every month	Alexandria
Shreveport	First Wednesday of every month	
Vernon	Third Thursday of every month	
Orleans	First Tuesday of every month	Shreveport
Ouachita	First Thursday of every month	

GRANTS FOR SURGICAL FELLOWSHIPS

The Woman's Auxiliary of the United States and Canadian sections of the International College of Surgeons has set aside $6,500 to be used for grants for surgical fellowships.

The announcement was made by Mrs. Walter C. Burket of Evanston, Illinois, national president of the Auxiliary.

The funds as voted by the Auxiliary's board of directors will be distributed as follows:

$3,000 to finance study abroad by an American or Canadian surgeon.

$3,000 to finance study in the United States or Canada by a foreign surgeon.

$500 for a senior medical student at the University of British Columbia, in Vancouver, B. C.

Details of the grants will be announced later.

WEST PALM BEACH INVITES YOU

The Southern District Meeting of the International College of Surgeons is to be held at West Palm Beach, Florida, in the Pennsylvania Hotel on January 29th and 30th, 1954. Surgeons and any other interested doctors of Louisiana are invited to attend the meeting.

The Pennsylvania Hotel is ideally situated on the west bank of Lake Worth and attractive rates have been established by the hotel for this meeting at the height of the Palm Beach social season, and in addition to the attractive entertainment there will be a top-level program featuring top-level surgeons. An informal dinner will be included in the reservation fee of $10.00. Outside events will be arranged for the ladies.

Reservations should be made early by writing The Manager, Pennsylvania Hotel, West Palm Beach, Florida, or Dr. Lloyd J. Netto, Chairman, 416 Comeau Bldg., West Palm Beach, Florida.

GASTROENTEROLOGISTS ELECT

At the Annual Meeting of the National Gastroenterological Association held in Los Angeles, Calif. on October 12, 1953, officers for the year 1953-4 were elected. The meeting was held on the opening day of the Eighteenth Annual Convention at which over 500 physicians from the North American continent were in attendance.

The presidency of the Association was assumed at the annual banquet on Tuesday evening, October 13 by Dr. Sigurd W. Johnsen of Passaic, N. J., who had been President-elect during the past year.

Elected officers for the coming year were: Dr. Lynn A. Ferguson, Grand Rapids, Mich, President-elect; Dr. James T. Nix, New Orleans, La., 1st Vice-President; Dr. Arthur A. Kirchner, Los Angeles, Calif., 2nd Vice-President; Dr. C. Wilmer Wirts, Philadelphia, Pa., 3rd Vice-President and Dr. Frank J. Borrelli, New York, N. Y., 4th Vice-President. Re-elected were: Dr. A. Xerxes Rossien, Kew Gardens, N. Y., Secretary; Dr. Roy Upham, New York, N. Y., Secretary-General and Dr. Elihu Katz, New York, N. Y., Treasurer. Dr. Samuel Weiss, New York, N. Y. was renamed as Editor-in-Chief of THE REVIEW OF GASTROENTEROLOGY.

The Nineteenth Annual Convention of the Association will be held in Washington, D. C. in October of 1954.

BLOOD PLATELET GROUPS IDENTIFIED: OFFERS NEW CLUE TO BLOOD DISEASES

Evidence that the blood platelets, little understood cells which help coagulate blood, can be grouped and typed like the red cells was presented for the first time before the American Association of Blood Banks, meeting in Chicago October 19th.

The importance of the observation, based on more than 50,000 tests, lies in the fact that the four groups and six types so far established are sometimes incompatible, like the four familiar red cell groups and the Rh and non Rh bloods. The discovery is therefore regarded as offering a key to certain types of blood disorders hitherto impossible or difficult to understand and treat.

The report was presented before the sixth annual meeting of the Association by Mario Stefanini,

M. D., for himself and Drs. Jyota B. Chatterjea, M. D., Gerald I. Plitman, M. D., William Damashek, M. D., and Mrs. Irma B. Mednicoff, Research Assistant, all of the New England Medical Center and Tufts Medical School of Boston, Mass. They emphasized that their results are preliminary and must be checked with caution.

ALLERGIES IN NEWBORN MAY BE MINIMIZED BY DIET RESTRICTIONS

Withholding cow's milk from the diet of a newborn child whose parents, brothers or sisters have a major allergy may prevent the infant from developing an allergy in later life, in the opinion of Drs. Jerome Glaser and Douglas E. Johnstone, Rochester, N. Y.

"Pediatricians and pediatric allergists have often observed that food sensitivities occur much more frequently in the first few months of life than in later infancy and childhood and in adults," the doctors wrote in the October 17 Journal of the American Medical Association.

"It is assumed that a physiological immunologic immaturity exists in the early months of life that results in sensitization and clinical symptoms caused by absorption from the intestinal tract of unaltered proteins in potentially allergic children."

As a result of this and similar observations, the doctors theorized that "if cow's milk protein, which constitutes the chief source of food and is the commonest cause of food allergy in the early months of life, were completely withheld from the infant's diet until this period of physiological immunologic immaturity passed, the symptoms of milk allergy might be minimized or even prevented."

VITAMIN C STRENGTHENS NETWORK FROM TEETH TO GUMS

Dr. Louis J. Baume, University of California dental research scientist, has reported the discovery of a fine network of vitamin C-needing tissues linking the teeth to the gums.

This connective tissue decays when vitamin C is deficient. Pyorrhea and other gum infections may result.

Dr. Baume states that these fibers are "protoplasm in chain form, arranged in bundles that run continuously from one cell into another, thus providing great strength and cohesion."

ASPIRIN REDUCED FEVER IN INFLUENZA AND COLDS FASTER THAN ANTIBIOTICS

Aspirin produced normal temperatures more speedily than the antibiotic drugs terramycin and erythromycin in patients suffering from such acute respiratory infections as influenza, bronchitis and the common cold, according to an article in the *Journal of the American Medical Association* (September 26, 1953).

Studies of the comparative effects of the three drugs on influenza-like diseases, which are believed caused by a virus rather than bacteria, are described in the medical publication by a team of U. S. Air Force doctors.

Their findings show that aspirin brought the patients' temperature down to normal in an average of 30 hours, compared with 41 hours for terramycin and 42 hours for erythromycin. The evidence indicates that the antibiotic drugs had no influence on the course of these diseases, nor was use of these drugs necessary since no secondary bacterial infections developed in any of the patients tested.

NEW RADIOPAQUE AGENT PROVIDES PRE-OPERATIVE BILE DUCT X-RAYS

Tests on more than 300 patients have demonstrated that the new contrast medium Telepaque is "an ideal substance for the study of the biliary tract," providing radiologists and investigating surgeons with "tremendously important" pre-operative views of the biliary ducts, according to Louis P. Divilio of the Department of Radiology, Grace-New Haven Community Hospital, New Haven, Conn.

A major purpose of the tests was to study the evacuation phase of the vesicle, he writes in *The X-Ray Technician* (Vol. 25:1, 1953.) Using multiple serial films, it was found that best visualization and optimum time for study of gall bladders filled with Telepaque was 10 to 12 hours following oral administration of the dye. Adequately visualized gall bladder density was also obtained in six hours. Films were made of patients in postero-anterior, left oblique prone and postero-anterior upright positions.

Gastric emptying was stimulated by means of an inexpensive fatty meal consisting of eight ounces of chocolate ice cream and four to six ounces of peanut oil mixed in a blender, the investigator notes. In many cases, ice cream with a high butter fat content also proved sufficient to cause contraction of the gall bladder so that the biliary ducts could be viewed.

Telepaque was well tolerated and produced fewer and less pronounced side reactions than other radiopaque agents, according to the study. Fewer cases of non-visualization were also encountered with the medium, which contains 66.68 per cent iodine, and it was determined that the right posterior oblique position provided better views of the ducts.

BOOK REVIEWS

Acute Peripheral Arterial Occlusion, by William D. Holden, M. D. Springfield, Illinois, Charles C Thomas, 1952, pp. 57. $2.35.

This monograph, which is one of the American Lecture Series, presents an excellent review of present day knowledge concerning acute peripheral arterial occlusion. The author concisely discusses diagnosis, etiology, pathological physiology, clinical manifestations, and management based on his own observations and well selected references from the literature. In considering the diagnostic phases of acute arterial occlusion, emphasis is placed on clinical observations rather than on elaborate instrumental studies. In his presentation of management, the methods recommended are sound. Not only of interest to surgeons, this little book could be read and referred to by general practitioners or internists who often see patients with acute peripheral arterial occlusion before they are seen by a surgeon.

AMBROSE H. STORCK, M. D.

Second Annual Report on Stress, 1952, by Hans Selye, M. D. and Alexander Horava, M. D. Montreal, Canada, Acta, Inc., 1952. pp. 526. Price $10.00.

This volume represents the second of a series of annual supplements to the book *Stress—The Physiology and Pathology of Exposure to Stress*, published by Selye in 1950. Its object is to serve as a guide to the entire literature on stress by compiling and sorting out the publications which appeared during the previous year. Since over 4,000 publications are listed, the task was a very arduous one. The report is written as a classified index of pertinent new facts with a concise evaluation of the principal findings. It is divided into three parts, preceded by an introduction.

In the introduction, the author attempts to evaluate the criticisms that have been levelled at the stress concept and cites recent experiments which would tend to support the general concept although leaving some question regarding specific details, such as the action of ACTH, and the involvement of the adrenals in rheumatic and other diseases. The first part presents references on the general physiology and pathology of stress, part II presents publications on special physiology and pathology of stress and part III is entitled "Afterthoughts" and presents a brief "sketch for a unified theory of medicine."

It should be emphasized that this is not a text monograph and certainly cannot be used as such. However, it should be invaluable to the investigator and clinician who is interested in the progress in the many and varied fields included in the concept of the general adaptive syndrome. The reader may, perhaps, question some of the statements and interpretations made in some of the introductory or summary remarks, but he will be grateful to the authors for having simplified the task of keeping abreast with the literature of this fast-moving field.

WALTER S. WILDE, Ph. D.

Rypins' Medical Licensure Examinations; edited by Walter L. Bierring. 7th ed. Philadelphia, Pa., Lippincott Company, 1952. pp. 856. Price $8.00.

This revised edition (7th) gives the examinee a firm footing on which to base his review of the basic sciences and clinical sciences. The material has been brought up to date and includes the fundamentals of the subjects treated. There is a tendency not to review the questions at the end of each section. For the benefit of those taking Florida State Boards the section on chemistry is inadequate, since one must be prepared for taking an examination on inorganic high school chemistry.

JAY STANGER, M. D.

Manual of Clinical Allergy; by J. M. Sheldon, M. D.; R. G. Lovell, M. D., and K. P. Mathews, M. D. Philadelphia, W. B. Saunders, 1953. pp. 413, illus. Price $8.50.

A very useful and practical text which brings together information not otherwise so readily accessible. Precise details of making extracts and the sundry allergic tests are given. The section on contact dermatitis is particularly helpful.

VINCENT J. DERBES, M. D.

Founders of Neurology; One hundred and thirty-three biographical sketches, prepared for the Fourth International Congress in Paris, by 84 authors; ed. by Webb Haymaker, M. D., with the bibliographical and editorial assistance of Karl A. Baer. Springfield, Ill., Charles C Thomas, 1953. Por. pp. 479. Price $10.50.

Since the history of a subject can only be the total of the contributions of workers in that field, this volume is indeed a source-book of the history of neurology. Contributions of 133 neurologists are described and documented by references to the literature and often by portraits. Wherever possible every effort has been made to relate the circumstances which led to the discoveries. The individual biographies are arranged in such a way as to give continuity in the history of the subject. The volume will be an invaluable reference book for the neurologist, the student, the historian, and the medical library.

MARY LOUISE MARSHALL.

The Journal of the Louisiana State Medical Society

Established 1844

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SUBSCRIPTION TERMS: $4.00 per year in advance, postage paid, for the United States; $4.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

THE JOURNAL OF THE LOUISIANA STATE MEDICAL SOCIETY

Formerly New Orleans Medical and Surgical Journal

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VOLUME 105

✦

JANUARY, 1953

through

DECEMBER, 1953

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